Investigation of a Congenital Defect Problem in Cow-Calf Herds

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Introduction

In March 1985 the manager of the largest cow-calf operation in Prince George, British Columbia presented one of us (CSR) with a disproportionately dwarfed, dead, newborn calf. He stated that he was seeing a disconcerting number of these calves for the second consecutive year and that he would like to know what was causing them. The calves tended to have normal sized bodies but short legs; they tended to be weak at birth; when they could stand they did so on hyperextended pasterns and had trouble ambulating on extremely unstable joints (Figure 1). By the end of the calving season we had seen more than 35 of these calves, most of which were dead or unmarketable.

FIGURE 1. A newborn calf affected with severe congenital joint laxity and dwarfism (CJLD). Laxity of the metacarpo- and metatarso-phalangeal joints results in their hyperextension so the calf's dewclaws contact the ground.

Two other ranches in the area had been experiencing a similar problem. The first ranch was devastated in 1984 when close to 60% of the calf crop was stillborn, dead within one month of birth, disproportionately dwarfed, or deformed (Figure 2). Owners of the second ranch reported that they had experienced these problems consistently since 1978, with up to 40% of their calf crop being affected in certain years.

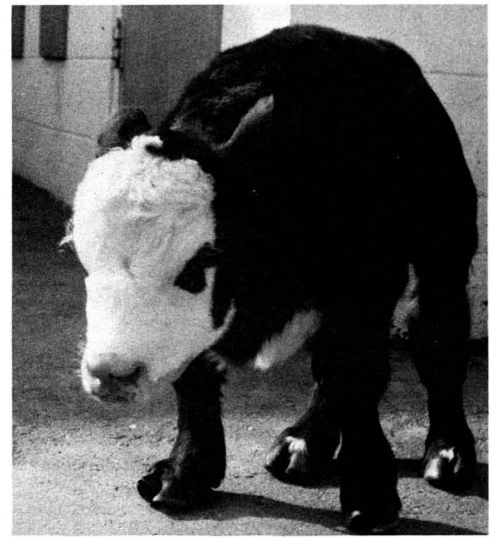

FIGURE 2. A CJLD calf showing marked dwarfism, metacarpo- and metatarso-phalangeal hyperextension, and mild superior brachygnathia.

Problems like this can be daunting in the day-to-day rigors of veterinary practice. By the time the abnormal calves hit the ground, the trail of clues is old and sometimes indistinguishable amongst a litter of poor records and foggy memories. Indeed, several previous attempts to elucidate the cause of the present problem had been unsuccessful. In this paper, we will outline our approach to solving this particular mystery, using basic descriptive epidemiologic methods and the active and interested participation of the affected ranchers. Theoretical considerations of developmental anomalies in farm animals in general have been presented previously by Rousseaux (1), and a detailed suggestion of how to define the etiology of congenital anomalies in general exists elsewhere (2).

Defining the Individual Incident

Our first step was to carefully define the problem (2). All dead calves from the affected ranches were necropsied and a detailed description produced of the clinical and pathological signs exhibited by the calves. The predominance of generalized joint laxity at birth, coupled with shortened limbs in 71% of the cases, led to our calling the anomaly syndrome "congenital joint laxity and dwarfism," or CJLD (3). *Lupinus polyphyllus* (Washington or large-leafed lupine) had been seen growing in the Prince George area and had been documented to contain the alkaloid anagyrine (4,5); ingestion of other lupine species containing anagyrine by pregnant cows from the 40th to the 70th day of gestation had been shown to cause a condition called "crooked calf disease" (6,7,8). However, the Prince George ranchers claimed their cows did not have access to the plant and a comparison of the clinical and pathological features of CJLD with "crooked calf disease" suggested the two conditions were not the same (9). Comparison of the CJLD condition with all the other congenital anomaly conditions of calves reported in the scientific literature uncovered a similarity with the "acorn calf" condition reported in the 1930's and 1940's in California (10,11), and late 1950's in Australia (12). The California researchers had shown the acorn calf condition was not hereditary, and was likely due to a maternal deficiency occurring between the third and sixth month of gestation (11). Unfortunately, the exact nature of the deficiency was never determined; furthermore, the Australian researchers suggested a simple deficiency was not likely the cause of the condition.

Descriptive Epidemiology

Having described the individual incident, we then set out to describe the patterns of occurrence, or epidemiology, of the CJLD condition. We attempted to **identify groups** of cattle on the various ranches differing in their management, sex, genetics, age, or location, and then determine the prevalence of the CJLD condition by group. Differences between groups could help us develop hypotheses concerning the cause of the congenital condition. By the time this phase of our investigation was fully underway, six Canadian cow-calf ranches had been affected by the condition. This allowed for comparison of group CJLD prevalence between ranches as well as within ranches. We also were able to search for similarities between ranches which might help explain the condition.

Individual calving records were examined and the ranch owners were interviewed. Our interview objectives were to obtain an overall understanding of ranch management, stock, and facilities; to record the movement of cattle from season to season; to clearly establish cropping and feeding practices; to determine what each ranch crew defined as an "anomaly" and how such occurrences were recorded; to draw out from ranch personnel theories of why the problem was occurring; to carefully establish when specific management or feeding practices were changed; and to determine if in any one year groups of pregnant cows were treated differently in some way from other groups on the same ranch. Fortunately, most of the ranchers kept relatively detailed calving records which, at the very least, contained accounts of the individual calving dates, the cow and calf identification, calf sex, and a remark concerning calf viability with the presence or absence of congenital defects noted. Most also had a reliable method of individually identifying cows and calves, usually by a combination ear tag and tattoo.

Genetic or Environmental

Only after defining the individual incident and establishing which calves born to which cows had the condition could we answer the following basic question: is the cause of CJLD primarily genetic or environmental? Genetic disorders occur in families (2), a pattern which did not emerge from our preliminary pedigree analyses. We documented the CJLD condition in nine cattle breeds on the six ranches, including Black Angus, Scottish Highland, Shorthorn, and Simmental. At one ranch, CJLD calves had been born to dams of eight and sires of seven different breeds. There was even documentation of a CJLD calf being born to a purebred Scottish Highland cow purchased in the United States and bred by artificial insemination to a registered Scottish Highland bull. This information, combined with the sudden dramatic appearance of CJLD with very high prevalence on certain ranches, suggested that CJLD calves were not a function of simple genetic inheritance or genetic mutation.

Prevalence and Descriptive Data

The results of our prevalence study can be seen in Table 1. The total prevalence varied considerably between ranches, from a low of 0% at RH in 1984 and 2% at BK in 1984, to a high of 46% at GF in the same year. First-calf heifers at GF and BH ranches consistently had more CJLD calves than did the mature cows (having had at least one previous calf). Significantly, we noted that on both these ranches the heifers were not fed separate from cows. However, at BK, where the heifers were fed separate from the mature cows over winter, and as a result received a small amount of grain (3 lb/head/day rolled barley) which the mature cows did not receive, the risk of CJLD for calves born to heifers was no greater than for cows. Also, at RH in 1982 when the heifers were supplemented some grain, their calves' risk of CJLD was infinitely smaller than the calves' of cows who did not get any grain. These findings led to our early hypothesis that supplementing over winter exclusive silage feeding with grain might reduce the risk of CJLD (Table 1).

TABLE 1. Prevalence of congenital joint laxity and dwarfism (CJLD) in the total herd, first-calf heifers, and mature cows at six Canadian cow-calf ranches.*

Ranch+	Year	Total Calves	Prevalence (No. CJLD/100 births)			Fed Apart**
			Total	Heifers	Mature cows	
GF	1980	107	26	50	20	no
GF	1981	108	22	44	18	no
GF	1982	114	17	50	10	no
GF	1983	68	15	46	10	no
GF	1984	110	46	58	37	no
GF	1985	101	23	26	21	no
GF	1986	48	33	–	–	–
BK	1984	751	2	6	0.4	yes
BK	1985	779	7	6	7	yes
BK	1986	665	6	6	6	yes
PM	1984	172	20	20	20	no
RH	1982	111	20	0	26	yes
RH	1983	120	8	10	8	no
RH	1984	120	0	0	0	yes
RH	1985	91	45	60	44	no
RH	1986	115	6	6	6	yes
HP	1987	97	33	–	–	–
BH	1986	73	22	53	11	no
BH	1987	59	29	75	17	no

* Note: stillbirths have been included in the figures for GF and HP, which had both experienced a notable increase in stillbirths since being afflicted with the problem: a small proportion of these may not have been true CJLD calves.

** Fed apart = heifers and mature cows fed separately overwinter?

\+ GF, BK, PM ranches were in Prince George, British Columbia; RH was in Ontario; HP was in High Prairie and BH in Barrhead, Alberta.

Feed

At all the affected ranches pregnant cows were fed silage (clover and grass silage) as the primary overwinter feed during the years calves with CJLD appeared. None of the affected ranches supplemented silage with hay overwinter during the years that CJLD was a problem; only BK and RH supplemented some grain, and only to the first-calf heifers (RH three years our of five: 1982, 1984, and 1986). Furthermore, the only year that GP and PM ranches experiences the problem followed the only winter silage was fed exclusively to the pregnant cows; hay (GP) or a combination of hay and silage (PM) had been fed during all previous winters. All other ranchers in the Prince George area fed wither hay or a combination of hay and silage to their pregnant cows overwinter. We had not been able to document the birth of CJLD calves at any of these ranches. It appeared likely that feeding a combination of hay and silage, rather than silage exclusively, somehow eliminated the risk of CJLD calves appearing.

Three very different silo types were used for silage production on the affected ranches, with a variety of base and side materials used in the horizontal trench silos (Table 2). None of the ranches used any silage additives, preservatives or acidifiers. All used plastic to cover the horizontal trench and heap silos except for HP, where a cover of fall rye was all that was applied to the top of the heap. Filling times and seal quality varied considerably between ranches. The dramatic differences in silo type and silage production method between ranches suggested that

no association existed between these factors and CJLD occurrence (Table 2).

TABLE 2. Silo construction type with base and side materials listed for each ranch affected with congenital joint laxity and dwarfism (CJLD).

Ranch	Silo Type	Silo Base	Silo Sides
GF	Horizontal trench	Dirt	Dirt
BK	Horizontal trench	Dirt and concrete	Wood
PM	Upright tower	–	–
RH	Horizontal trench	Concrete	Concrete
HP	Heap (packed)	Dirt	Round straw bales
BH	Heap (packed)	Dirt	Round straw bales

A variety of mineral mixes (or none at all) were used at the affected ranches, suggesting that mineral supplementation was not an important factor (Table 3). Importantly, no herbicides were used at HP (Table 3). Finally, on all places the birth of CJLD calves was not clumped at any time during the calving season: CJLD prevalence was fairly constant throughout. It was not possible, therefore, to implicate a "point-source" fetal insult (Table 3).

Natural Experiments

We were fortunate to discover two natural experiments in the data, one at GF ranch and one at PM ranch. Natural experiments occur when, by circumstance, two subgroups within the herd, which were treated identically except for one or two risk factors, can be identified in the records (2). In both natural experiments a group of pregnant first-calf heifers was purchased in late fall and brought to the respective ranches. This allowed comparison of the purchased group of heifers with those that had been on the ranches since their birth.

In November 1983, 16 bred heifers purchased in Edmonton were brought to the GF ranch; 13 of these heifers calved the following spring. That same spring of 1984, 32 home-bred heifers calved. Notably, while 53% of the home-bred heifers had CJLD calves, none of the purchased heifers had any CJLD calves. That same November, 34 bred heifers were purchased at the auction market in Vanderhoof, British Columbia and brought back to the PM ranch. In spring, 38 home-bred PM heifers calved. Unlike the GF situation, at PM both the home-bred and purchased heifers had similar prevalences of CJLD calves and stillbirths, 47% and 50% respectively. The PM purchased heifers came from a group of 170 bred heifers in Vanderhoof, the remainder of which, once tracked down by district agriculturist Garth Elgie, were reported to have had no similar problems. A more general survey of the Vanderhoof area also resulted in no similar problems being found.

Taken together, the results of the two natural experiments seemed contradictory. Had something different occurred on the two ranches which could account for the apparently contradictory findings? Further investigation uncovered an important difference. At PM ranch, the purchased heifers were mixed immediately with the 38 home-bred heifers and fed silage exclusively from mid-December to calving, with no supplementation of grain or hay. However, at GF ranch the 16 purchased heifers were kept separate from the rest of the herd and supplemented with 2 lbs/head/day rolled barley from January until they calved. The home-bred or "keeper" heifers received no such supplement.

TABLE 3. Tabulation of other risk factors that might be associated with CJLD at the various ranches: mineral mix, herbicides, fertilizers, and method of breeding.

Ranch	Mineral Mix	Crop Herbicides	Crop Fertilizers	Breeding Method
GF	Biophos & salt	None	Variety	A.I. & Natural
BK	Complete	Variety	Variety	Natural
PM	None	–	–	Natural
RH	Complete	Variety	Variety	Natural
HP	Iodized	None	None	Natural
BH	Complete	–	–	Natural

Note: (–) means information unavailable.

Now the findings appeared explicable. The similar prevalence of CJLD calves in the two PM groups suggested

the insult to the fetus occurred sometime over winter, after the arrival of the Vanderhoof heifers on the ranch in November. Our inability to document similar problems in bred heifers that spent the winter in Vanderhoof supported this hypothesis. Perhaps by supplementing the GF purchased heifers with grain, the GF calves were not exposed to the CJLD insult. This gave further credence, then, to our theory of an overwinter teratogenic insult resulting from feeding silage exclusively without supplementation of grain or hay to pregnant cattle.

Conclusions

We derived the following conclusions or hypotheses from the results of our descriptive investigation. First, the insult to the fetus occurred over winter, probably between early December and calving. Second, all the affected ranches fed red clover and grass silage exclusively over winter to their mature pregnant cows. Third, ranches which fed hay in combination with silage have not been documented to have the problem. And finally, supplementing the clover/grass silage with grain (2-4 lbs/hd/day rolled barley) appeared to reduce the prevalence of CJLD, although this feeding practise did not completely eliminate the problem.

Summary

By carefully working through the investigative stages of defining the individual congenital anomaly, looking through the calving records and interviewing the ranchers to describe the epidemiology or patterns of occurrence of the congenital syndrome, "asking the data" whether the syndrome likely had a predominantly genetic or environmental cause, and identifying two natural experiments, we developed testable hypotheses concerning the cause of the congenital syndrome, which we termed congenital joint laxity and dwarfism or CJLD. Confirmation of the hypotheses generated by this descriptive study was achieved by conducting feeding trials at three of the affected ranches during two subsequent years (3).

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