Interrelationships among Production and Clinical Disease in Dairy Cattle: A Review

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Abstract

Epidemiological evidence is presented in order to answer two questions. The first question is: "Does high milk production put a cow at increased risk of disease?" The answer to this question seems to be "maybe" for milk fever, but "no" for most other common diseases (veterinary-assisted dystocia, retained placenta, metritis, cystic ovary, ketosis, left displaced abomasum, and mastitis). The second question is: "Is low milk production a consequence of disease?" For most diseases the answer is a cautious "yes".

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Introduction

It seems reasonable to ask whether the continuing increases in the level of milk production of our dairy cows will result in increased levels of disease. It won't do to compare current incidence rates of disease to rates from years past; records aren't that reliable, and management skills have changed considerably. A better way of exploring the question is to compare rates of disease among current higher-yielding cows to those in lower-yielding cows, especially under similar management.

A related question concerns the cost of disease. Surely, clinical disease is undesirable because of treatment costs, but are there also costs related to decreased milk production following disease?

The purpose of this paper is to review the epidemiological literature in order to answer these two questions:

Question 1 (Q1): Does high milk production put a cow at increased risk of disease?

Question 2 (Q2): Is low milk production a consequence of disease?

Materials and Methods

Several limitations were placed on this review. First, the literature reviewed was primarily that of veterinary medicine and epidemiology — some studies from the animal science literature have been included, but others may have been missed. Second, papers were excluded if the data were from university herds or other herds maintained for research purposes. This review is intended to be pertinent to managers and veterinarians of private commercial herds, whereas the management of research herds may have very different goals and conditions. (Consider, for example, possible differences in policies regarding culling for low production.) It should be noted, however, that restricting the studies to those of private herds meant that the studies were observational rather than of the intervention type — it would be unethical to impose disease or low production on private farms just to create conditions for answering research questions.

The third restriction was that studies were excluded from the review if the data were based on herds rather than on individual cows. Knowing that rolling herd average is related to incidence rate doesn't mean that the higher-producing cows in the herd were the ones who got sick (an example of "ecological fallacy"). The fourth restriction was that studies were useful only if milk yield data were clearly for times *before* or *after* the cow was sick. Milk yield *while* the cow was sick won't help answer either Q1 or 2, both of which require a clear time sequence (not concurrence) in events.

The final restrictions concern the diseases included. This review is limited to clinical disease only, which excludes studies of, for instances, somatic cell counts or ketonuria. In addition, the only diseases included are veterinary-assisted dystocia, retained placenta, metritis, cystic ovary, clinical milk fever, clinical ketosis, left displaced abomasum, and clinical mastitis.

The 15 papers included in this review are summarized in Table I. One thing to notice about these studies is that except for references 1 and 10, all are from this decade. Epidemiology is a relatively new discipline; large-scale detailed data sets and scientists with the skills (and interest) to analyze such sets are fairly new, also. Another thing to notice is that most of the studies *are* large-scale . . . they include hundreds or thousands of lactations. Such large numbers increase the expenses of collecting and analyzing the data, but there are important advantages. Large numbers often mean several herds were included, so that the conclusions are less likely to depend on the peculiarities of a single herd (therefore, the conclusions generalize more readily to other herds). More basically, though, large numbers are needed in order to detect many differences that are of practical interest (or, to be confident that such differences don't exist). For instance, it takes (in very round numbers) 500 lactations *per group* to tell that an incidence rate of 5% is different from 10%, or that

TABLE I
Summary of Articles Reviewed

Reference	Location	Sample Size	Control for Counfounding Variables	Measures of Milk Yield[a]
1	Ontario	82	no	yield/d prior to diagnosis
2	Ontario	82?	yes	previous lactation 305 d yield and BCM; BCM
3	Ontario	810	yes	previous lactation BCM; BCM
4	Ontario	2,875	yes	previous lactation BCM
5	Ontario	2,875	yes	BCM, yield/day of calving interval
6	New York	2,190	no	estimated transmitting ability for milk
7	New York	1,983	yes	estimated transmitting ability for milk
8	New York	1,374	yes	previous lactation ME
9	New York	2,850	yes	estimated transmitting ability for milk, previous lactation ME; 305 d ME; 305 d ME
10	Virigina	591	no	previous lactation 90 d yield and 305 d ME
11	Quebec	23,873	yes	90 d, 305 d, yields
12	France	343	no	1st test d yields
13	Finland	8,201	yes	4% FCM of the latest 12 mo before calving
14	Finland	51,449	yes	previous lactation yield
15	Israel	695	yes	milk yield of the latest 120 d before calving

[a] BCM = breed class average for milk; ME = mature equivalent milk yield; FCM = fat-corrected milk

one group produces 250 kg (assuming about an 8400 kg mean production and about a 1400 kg standard deviation) more milk in a lactation than another group.

Whether or not the study included control for confounding variables also is indicated in Table I. The confounding variables controlled typically included at least herd, breed, age/ parity, and season; often control for possible confounding was considerably more intensive. Methods of control for confounders ranged from matching to calculating deviations from herdmate averages to adjustment for the confounding variable in the multivariate statistical analysis.

Studies 1 through 5 were done by an interrelated group of scientists at the Ontario Veterinary College, but the studies used four different, independent data sets. In contrast, the four papers from the New York State College of Veterinary Medicine present an evolving series of analyses of data from the same data set (but of data collected over different periods of time). Because of the common underlying data collection project, two New York papers should not count in the review as heavily as two totally independent papers (say, one from Ontario and one from Israel). Finally, all studies were of Holstein-type cattle except for 13 and 14, which were of Finnish Ayrshires.

The measures of milk yield or milk yield potential (estimated transmitting ability; a measure based at least in part on pedigree) that were used in the analyses are listed in the final column of Table I. References 1-4, 6-10, and 12-15 used measures from before the cows' diagnoses (both pedigree and previous lactation yield are fixed before the current lactation begins); these studies, therefore, are pertinent to Q1. (Reference 12 was included because the only disease of interest, metritis, was limited to diagnoses later than 30 days postpartum and the first milk test usually occurs earlier than this.) There may be confounding by length of dry period in studies 13 and 15, because the "12 mo" or "120 d" preceding calving include the dry period, and dry period length did not seem to be controlled in the analyses. Very long (or short) dry periods would, therefore, decrease (or increase) the measure of previous milk yield, and might also influence risk of disease. (For instance, a cow might become overconditioned in a long dry period . . . and then have difficulty calving.)

Similarly, references 2, 3, 5, and 9-11 are pertinent to Q2. Unfortunately, in none of these studies was there a clear separation between the period of clinical disease and the period of milk recording. The diseases under consideration occur most commonly in the early stages of lactation, though, so *most* of the milk recording period will have been subsequent to the time of diagnosis. (This argument will be somewhat poorer for metritis, cystic ovary, and mastitis than for the other diseases.)

Results and Discussion

Question 1

The results of the literature review for Q1 (does high yield increase risk?) are summarized in Table II. For all the diseases except milk fever, left displaced abomasum , and mastitis, there is a preponderance of references in the "no" column. It was surprising to find only a single study which examined Q1 for mastitis. However, several potential papers were eliminated from the review because *sub*clinical mastitis (intramammary infection or somatic cell count) was the object of the analysis, or because the data were based on herd-level rather than on cow-level measurements.

Left displaced abomasum presents a small problem with two papers saying that high yield was a risk factor, and two saying it wasn't. I believe that the weight of evidence is stronger on the "not a risk factor side" for these reasons: references 4 and 8 were collected prospectively (intentionally for these uses by the scientists involved), are from more recent lactations, have considerably larger sample sizes, and involved broader and more sophisticated control for potential confounding variables.

Milk fever is the only disease of the eight reviewed in which high milk production might be a risk factor or clinical disease. Reference 7 (based on pedigree) and 8 (based on previous mature equivalent milk yield) tend to cancel each other out because they're based on the same data project. However, that still leaves the excellent study from Ontario (4) and the massive data set from Finland (14; even though the Finnish cows were Ayrshires rather than Holsteins).

In summary, most studies suggested that high milk production or milking potential was not a risk factor for clinical disease (with the possible exception of milk fever). However, the limitations in the evidence should be understood. It's important to remember that, except for references 1 and 12, none of the "before diagnosis" measures of milk production were from the *current* lactation. These analyses cannot rule out the possibility that a cow was "outdoing herself" just before she became diseased (that is, performing better in the current lactation than would have been expected based on pedigree or previous performance). This possibility can be ruled out for dystocia and retained placenta (and probably for most cases of milk fever and left displaced abomasum, because typically the occur so soon after calving). However, the possibility should be explored for the other disorders — especially for those cases of metritis, cystic ovary, ketosis and mastitis that occur well into the lactation.

Other limitations also exist: the data are observational and cannot *prove* causation, and there is the possibility of confounding in several studies (especially, as mentioned previously, in references 1, 6, 10, 12, 13, and 15). The last limitation that will be mentioned is that analyses typically did not correct for disease in the previous lactation. It is possible that some of the diseases (especially cystic ovary, milk fever, and mastitis) are repeatable. If disorders do tend to recur in succeeding lactations *and* if the disorders do result in subsequent losses in production (see Q2), then failure to account for previous disease could bias against discovering an association between high milk yield and subsequent disease.

Question 2

Limitations in the reference cited for Q2 have been mentioned previously in this review, including potential confounding (10), inability to prove cause-and-effect in observational studies, limited sample size (?: 2, 3, 10), and failure to separate completely the disease period form the milk measurement period. An additional point must be made which is crucial to my interpretation of the evidence: the difference between kg/calving interval day (reference 5) and standardized 305 d production (breed class average or mature equivalent). In my opinion, kg/d is the superior measurement because it relates the *entire* lactation production to the *entire* period of time during which the cow must be fed, housed, bedded, and depreciated. In contrast, 305 d milk, even if adjusted for days open, does not account fully for length of dry period or for shape of the lactation curve after 305 d. It is important that Dohoo and Martin (5) acutally used *both* measures of milk production in their analyses, and showed that the measures can lead to opposite conclusions. That is, in several instances a predictive variable had a *positive* effect on 305 d yield but a *negative* effect on kg/d (5). Because I believe that kg/d is the superior milk yield measurement, the results in reference 5 that were based on 305 d yield were ignored, and evidence from reference 5 was given greater weight than evidence (based on 90 d or 305 d milk) from the other studies.

The summary of the literature review regarding low milk yield as a consequence of disease is in Table III. If evidence suggested that there was an effect of clincial disease on production, then the suggested effect

TABLE II
Studies Giving "Yes" or "No" Answers to Question 1:
"Does High Milk Yield (or Potential) Increase Risk of Clinical Disease?"

Clinical Disease	Reference Number of Study	
	Answer = "Yes"	Answer = "No"
Veterinary-assisted dystocia	—	3, 4, 8
Retained placenta	—	3, 4, 8
Metritis	—	3, 4, 12[a], 15
Cystic ovary	9	3, 4, 6, 10
Milk fever	4, 7, 14	8
Ketosis	14	4, 8, 13
Left displaced abomasum	1, 2	4, 8
Mastitis	—	4

[a] Metritis restricted to cases > 30 d postpartum

TABLE III
Studies Giving "Yes" or "No" Answers to Question 2:
"Does Clinical Disease Result in Lowered Milk Yield?", and Estimated Effects

Clinical Disease	Reference Number of Study		Probable Effect
	Answer = "Yes"	Answer = "No"	
Veterinary-assisted dystocia	11[a]	3, 9	−5.3 to −8.8%[a]
Retained placenta	5	3, 9	−0.4%
Metritis	5	3, 9	−2.0 to −4.6%
Cystic ovary	5	3, 9, 10	−2.4%
Milk fever	5	9	−0.5%
Ketosis	—	5	0?
Left displaced abomasum	2, 5	—	−1.4 to −9.8%
Mastitis	9	5	0?

[a] Surgical dystocia associated with stillbirth; otherwise, no effect

(as a % of production) was listed. The conclusions are less clear for Q2 than they were for Q1, except for veterinary-assisted dystocia and left displaced abomasum. Both disorders have > 1 paper indicating consistent conclusions, and both disorders occur so early in the lactation that yield can be interpreted as entirely subsequent to onset of the disorder. There seems to be a severe cost (5.2 to 8.8% of yield) of dystocia involving a surgical delivery of a stillborn calf (11; Table III), but veterinary-assisted dystocia is not otherwise associated with decreased production. (It still would be nice to have this confirmed using kg/d.) The other "surgical" disease reviewed — left displaced abomasum — also carries a penalty in lowered milk yield (a loss of 1.4 to 9.8%).

For the other diseases, the evidence is less clear regarding Q2. Only a single (albeit excellent) study examined the effects of clinical ketosis; confirmatory studies are needed. The greater evidential weight given to reference 5 counterbalances the "no effect" conclusions of other studies for retained placenta, metritis, cystic ovary and milk fever. Additional studies regarding the production effects of these diseases also are needed. The additional studies should use the kg/d of calving interval type of measurement, but for metritis and cystic ovary should pertain to the lactation and calving interval *after* diagnosis. The studies for retained placenta and milk fever will have to be especially large, because the effects suggested by Dohoo and Martin (5) are small. (Alternatively, effects of <0.5% could be declared "trivial", and efforts to define production losses could be concentrated on disorders other than retained placenta and milk fever.)

The results regarding clinical mastitis are, at first, surprising: there are only two pertinent epidemiological studies, the results are conflicting, and the one with the better measurement of milk production indicated no associated loss of production. In contrast, the dairy science literature indicates that *sub*clinical mastitis *is* associated with decreased yield. Dohoo and Martin speculated (5) that their results might be explained by the fact that their analysis of the effect of clinical mastitis on kg/d controlled for somatic cell count level, and that treatment (clinical cases) might be protective compared to lack of treatment (subclinical cases). Clearly, more work in this area is needed.

In summary, it seems reasonable to conclude that left displaced abomasum and surgical deliveries of dead calves are associated with losses of milk production, but that other forms of dystocia probably do not decrease yield. Additional studies are needed to answer Q2 for the other diseases, but there are suggestions of lost yield as a consequence of milk fever, retained placenta, metritis, and cystic ovary; the losses might be important for the latter two diseases.

Conclusion

The best evidence currently available suggests that the cow who produced more milk than here herdmates is not at increased risk of any disorder other than milk fever. This should be a reassuring finding to dairymen, because milk fever may be the one disorder out of the eight reviewed that is most readily prevented; preventive dry period management could be targeted to the high-yielding cows in the herd. However, a series of studies that examine *current* lactation milk yield prior to diagnosis of metritis, cystic ovary, ketosis, and clinical mastitis are needed to increase confidence that high milk yield is not a risk factor for these four disorders.

Additional, even more sophisticated, studies also are needed to settle the question of whether retained placenta, metritis, cystic ovary, milk fever, ketosis, and clinical mastitis are risk factors for lower milk yield. However, there is little evidence suggesting that lower yield does follow retained placenta, metritis, cystic ovary, and milk fever, and stronger evidence indicating production losses following left displaced abomasum and surgical delivery of stillborn calves. Other forms of veterinary-assisted dystocia, however, are not risk factors for decreased milk production.

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