PEER REVIEWED

High Production and Health–A Curious Paradox

John Fetrow, *VMD, MBA*
College of Veterinary Medicine, University of Minnesota, St. Paul, MN 55108
Steve Eicker, *DVM, MS*
Valley Agricultural Software, King Ferry, NY 13081

Abstract

While there is considerable belief that high producing cows are more likely to suffer various negative events (disease, culling, etc.) than low producers, there is very little data to support that belief for most conditions. With the possible exceptions of clinical mastitis and milk fever, most studies find no causal connection between high production and the risk of negative events in a cow's life.

Résumé

Bien que l'on croit généralement que les vaches qui produisent beaucoup de lait soient plus sujettes à des problèmes (maladie, réforme, etc.) que les vaches en produisant moins, il existe bien peu de données pour étayer l'hypothèse dans la plupart des conditions. À l'exception possiblement de la mammite clinique et de la fièvre du lait, la plupart des études ne trouve pas de relation causale entre la forte production et le risque qu'une vache développe des problèmes durant sa vie.

The Paradox

There is a paradox that pervades dairy farming, a paradox not often spoken about in simple terms, but one that shapes many decisions made by dairymen and those who advise them.

1. On one hand, it is self-evident that if dairymen find ways to remove stressors from cows, cows will respond with improved health and produce more milk. Dairymen work hard to provide balanced rations, keep cows comfortable, cool cows in hot weather, dip teats and vaccinate to prevent disease, ensure adequate ventilation and provide clean accessible water. All these things are done to minimize stress for their cows. It is generally accepted that if these things are done well, there will be less disease, fewer "broken" cows and less need to replace cows (cull) that have been damaged to a point of low economic value. In short, it is generally accepted that reducing stress results in less risk and higher milk production.

2. On the other hand, there is also a strong belief that high-producing cows are more stressed than their low-producing herd mates. High-producing cows are believed to be at greater risk of clinical disease and other negative consequences of high production that could ultimately cause the cow to "crash" in production or to be culled (or die). Dairymen commonly believe that high production is associated with poor reproductive performance rates, more mastitis, metabolic and other disease, and higher culling. High-producing cows are described as being "stressed", "walking a tightrope", or "on the edge". Dairymen express concerns about "burning out cows" and talk of not "pushing their cows" for higher production. The short version of this belief might be termed "high production breaks cows".

The paradox is obvious: if cows with lower stress become higher producers, how can it be true that high production stresses and "breaks" cows?

Consider the first part of the paradox. There is an enormous array of science to support the notion that as we provide better care for the cow (feed, environment, disease control, comfort, etc.), she responds by consuming more feed and producing more milk. As we remove the "bottlenecks" (stressors) that inhibit her from expressing her true genetic potential, the cow is "freed" to improve milk production and suffers less disease. It certainly makes sense that higher-producing cows should be healthier and less stressed.

At the same time, it is equally obvious that cows are sometimes hurt by management actions intended to increase production. An obvious example is overfeed-

ing grain with the goal of increasing milk production. In the short term this strategy might be successful, but in the long term the effects on foot and rumen health make it counterproductive. In this example, it is not the high production *per se* that leads to the cows' problems, but an error of management. If cows achieved higher production by another route (increased access to water, for example), there is less likelihood of negative health effects, or even improved health, accompanying higher production.

Avoiding Logical Leaps Over the Cliff of Causality

If there is a wealth of support for the first part of the paradox (reducing stress increases production), what is the source of the belief in the second, contradictory part? We assert that the second part of the paradox comes largely from the general observation that as the dairy industry has changed over the past several decades, increased production per cow has been accompanied by notable increases in certain types of problems. With the two observations (more milk, more problems) appearing concurrently, it is natural to believe that the two are linked, and that increased production causes an increased level of certain problems. Many people falsely conclude that because certain clinical problems have increased as production levels have increased, an individual cow is more at risk as her production level increases. There is little data to support this, but it is widely believed, and in fact may be the prevailing assumption across the industry.

Consider an example of the changes in the dairy industry over the past several decades. Figure 1 shows Minnesota annual summary data for herds from 1950 to 2001; 50 years of history.[16] It shows a clear association between increased milk production of cows on Dairy Herd Improvement Association (DHIA) test and the cull rate, as measured by the proportion of cows that exited the herd each year. Statistically, the correlation between culling and production is incredibly tight (R^2 of 0.88, p <<<.01). Does the correlation prove that high-producing cows are more likely to be culled? Does this make sense? Do most dairymen choose to cull more high-producing cows than low-producing cows? In fact, both common sense and published papers show that higher production protects cows from being culled.[10] Viewed from the other perspective, does the high (and statistically significant) correlation prove that increasing culling increases production? Or as a final interpretation, does this graph only establish that increased production and increased culling happened at the same time? By itself, a causal connection cannot be proven.

In epidemiology, falsely concluding that two events occurring simultaneously must be causally connected is referred to as an "ecological fallacy". Miles traveled by air in the United States have gone up steadily over the past several decades, as have the number of white tailed deer killed on highway. Few would consider these concurrent trends to be causally connected. Similarly, after a little thought, most of us would remain skeptical that Figure 1 proves that increased milk production makes a cow more likely to be culled.

But consider Figure 2, which presents Minnesota DHIA data for the past two decades, showing milk per cow and average days open. Again, there is a tight correlation between the two parameters (statistically significant correlation, $R^2 = 0.80$, $p<0.0002$). What does this correlation mean? Actually, it means no more than the correlation of culling and milk production did (or air miles and dead deer). It only means that these two things happened at the same time. Unfortunately, nearly everyone in the dairy industry leaps to the conclusion that Figure 2 (and the accompanying statistical testing) proves that higher milk production causes poor reproduction. It is just as easy to conclude that poor reproduction causes higher production, and to start

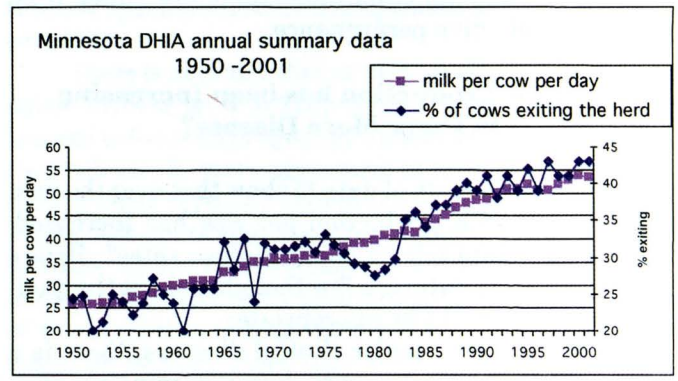

Figure 1. Minnesota DHIA: production per cow and percent of herd culled per year.

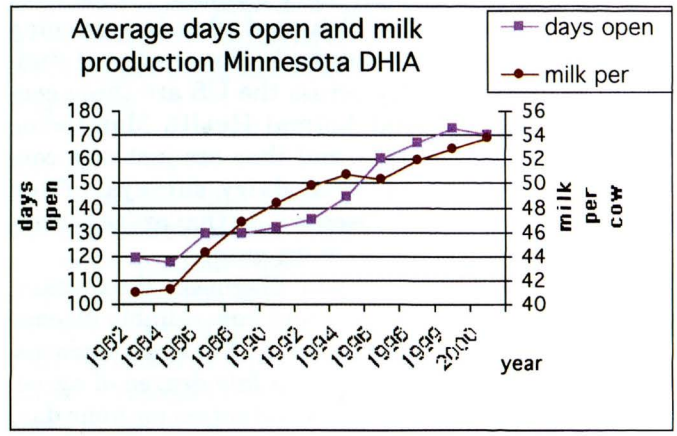

Figure 2. Minnesota DHIA data: days open and milk per cow.

breeding cows when they are not in estrus to increase milk output. There might be a causal connection between increased milk production and declining reproduction rates, but Figure 2 does not prove it.

Finally, even if an association were causal for the industry as a whole, it does not mean an individual cow with higher production would necessarily be more at risk of, in this case, poor reproduction. What is true as a dynamic across a population does not necessarily extend to the individual.

Consider a non-dairy example. Over the past several decades, people in the US have been more at risk of obesity and the attendant illnesses that go along with obesity. It is also true that we have become more affluent as a society over the same period. Finally, it is probably true that the two (affluence and obesity) are causally connected for the population as a whole. This does not mean, however, that a person winning the lottery would be expected to become obese. This non-dairy example makes sense to most people, but doesn't stop many in the dairy industry from making the false logical conclusion that if reproduction has become poorer while milk production has increased, it must be true that high producing cows must therefore suffer from poorer reproductive performance.

We Know Production has been Increasing. Is There More Disease?

There are plenty of data to show that over the past five decades milk production per cow has increased. What do the data tell us about disease rates? Before looking at the literature, a few thoughts about the quality of the data might be appropriate.

First, for most major clinical diseases, there is a remarkable paucity of quality data regarding the incidence of the disease. Much of the data are derived from university studies where the researcher collected data on a convenience sample of herds, typically university herds or cooperating local dairy farmers. These data may not represent a fair picture of what is happening across the industry. The only truly cross-sectional studies of the dairy industry across the US are those conducted by the National Animal Health Monitoring System (NAHMS, USDA), and they are just now conducting the second national dairy surveys. Even NAHMS must deal with disease data that are producer-reported, and subjective in many cases.

Second, the issues of who diagnoses the disease, the definition of the disease, and how reliably disease is recorded all must be considered. For some diseases, e.g. retained placenta, there is a fair degree of agreement of what constitutes a case (retention past one day) and the diagnosis is fairly obvious. What about ketosis? Was each cow's urine, milk, or blood checked daily in the post-partum period? What test was used and how sensitive and specific was the test? Who made the diagnosis? How ketotic must the cow be to count as a case? How reliably was it recorded? It is obvious that studies of the incidence of ketosis will be fraught with problems of inconsistency across even well conducted studies.

What are other reasons disease might seem to be associated with production? Systematic bias may be introduced into the data, particularly by deliberate culling decisions. Low-producing cows with repeated bouts of mastitis might be culled, while high-producing cows might be kept. After culling low-producing cows with mastitis, a survey of the herd would show an association between high production and mastitis.

Some perceived associations might be faulty when we observe larger populations of cows. A 4% death rate in a 50-cow dairy means that two cows die each year. Those two deaths are easily "explained away" as unusual, exceptional events, not indicative of any particular causality. The same 4% death rate in a 1,000-cow dairy equates to 40 cows, or nearly one each week. This apparent problem begs for an explanation, even if it may be a fabricated one. Finally, our record systems and our ability to capture accurate and complete data have been steadily improving. What appears to be an increased incidence of a problem may be nothing more than increased quality of data recording.

With these difficulties firmly in mind, it is still interesting to consider what we know about the rates of disease and the association or connection of disease and milk production over the past several decades. If there was evidence that the rate of a disease had increased during the period when production also increased, then one could form a hypothesis that increased production might lead to more of the disease. The association would not be proof, but could stimulate more reliable testing of the hypothesis by other sorts of studies.

Left Displaced Abomasum

Left displaced abomasum (LDA) is a fascinating problem in the dairy cow. Before the 1950s, the disease was simply not known. The 1916 USDA compendium of cattle disease[20] lists most of the digestive diseases we know today, and a few we would not often diagnose: bloat (acute and chronic), feed overload, hardware (reticuloperitonitis), hair concretions, vomiting, indigestion or dyspepsia. Notably, the book makes no mention of displacement of the abomasum. While it is possible that veterinarians simply overlooked the disease, it seems implausible that no surgeon or pathologist cut open an ill or dead cow and discovered the abomasum was in the wrong place. It seems far more reasonable to conclude that the disease didn't exist in 1916.

The first documented cases of LDA in the English literature were reported in England in 1950,[8] followed swiftly by other reports.[1] Still, the disease was quite rare and prompted American reviewers of the condition in 1954 to open their paper with "Displacement of the abomasum is one of several obscure conditions that are of chief importance because they cloud and confuse diagnosis of digestive and metabolic diseases in cattle practice."[17] Less than a decade later, the authors of a series of 80 cases opened their paper quite differently: "Perhaps one of the most remarkable features of dairy practice in the last decade has been the apparent marked rise in the incidence of displacement of the abomasum of the dairy cow."[19] When first reported by Begg[1] (1950) and Ford[8] (1950), there was no indication that within a few years this condition would be one of the most common surgical conditions of the bovine alimentary tract.[19] In some respects, the sudden appearance of the disease mimicked the introduction of a new infectious pathogen into a susceptible population.

Later surveys of the incidence of LDA in herds have shown a fairly wide variation. A Canadian survey of 32 Ontario herds reported an incidence of LDA of 1.2%.[3] An early state level NAHMS survey in Ohio reported an incidence of 8.4%,[15] while similar state surveys in California[11] and Michigan[12] did not report LDA as a category (too few cases). A 1995 report from New York cited an incidence of 6.3% in 25 herds with 8,000 cows.[9] The national 1996 NAHMS dairy study reported an incidence of 2.8%.[21] Since these were owner reported cases, the incidence is probably a conservative estimate of the true rate.

While the rates may differ from one study or decade to the next, it is clear that LDA is now a common disease of dairy cows, a very different situation than existed 50 years ago. While it is possible that some diagnoses were missed before the development of simultaneous auscultation and percussion (pinging), at least some observant pathologists should have found an abomasum in the wrong place if the disease were actually occurring. Something has fundamentally changed that puts the cow at more risk of LDA, whether management (feeding, feeds, housing, etc.) or the cow herself.

Reproduction

General performance

There appears to have been a distinct shift over the past two decades in reported overall reproductive performance in dairy cows. The average days open in Minnesota dairy herds has increased from 120 to 170 days (Figure 2). Figure 3 shows the data for average conception rate in pregnant cows.[16] In the past two decades, conception rate in pregnant cows has dropped from 58% to 48%. Some of these shifts may reflect

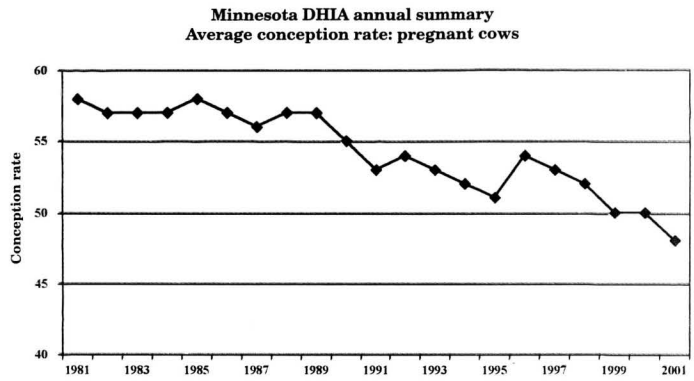

Figure 3. Minnesota DHIA data: average conception rate in pregnant cows.

changes in the behavior and policy of the producers themselves in terms of how long they are willing to breed open cows. Some of the change may reflect improvements in record keeping and more accurate reporting of the raw data through DHIA systems. As much as we would like to use these data to prove a fundamental shift in reproductive performance on dairies, we only have a correlation. An associated question is whether the incidence of specific reproductive diseases has also changed over time.

There is no doubt that as milk production increases, dairy farmers tend to keep cows longer. As they have learned to focus on pregnancy instead of conception rate, they will also be more aggressive at breeding more cows, even if their reported reproductive performance looks worse. Ten years ago, many farms did not have on-farm software, and it was common for less progressive farms to only report the cow's final breeding to DHIA. Much of the apparent change in reproductive performance might be nothing more than better data recording on better farms. Particularly in the past decade, there has been an increase in the use of synchronized and timed breeding programs. Effects on first service conception may not reflect any real change in the cow's basic physiology. As these practices have changed and we have fewer cows that conceived on their first breeding, it appears that first-service and overall conception rates are decreasing.

Metritis

With the data available, it is difficult to decide whether there has been a shift in the incidence of metritis. In a Canadian report in the late seventies, the incidence of metritis was 18.2%.[3] Early NAHMS state surveys reported an incidence of 35%[15] and 7%.[11] Such wide variation in reported incidence may reflect differences in case definition and reporting, not true differences in the disease itself. In a later New York survey, metritis incidence was 7.6%,[9] and when using a different set of dairies in 1998 it was 4.2%.[10]

Retained placenta

The reported incidence of retained placenta shows much less variability than metritis. In a Canadian study, the incidence was 8.6%.[3] State NAHMS surveys reported 8.0%[15] and 4.7%,[11] while surveys in New York showed 7.4%[9] and 9.5%.[10] The national NAHMS survey reported 7.8%.[21] The case definition of retained placenta is clear and the problem is easily seen, making the data from surveys more reliable than for other diseases. At least in the last two decades, the average rate has been fairly consistent at roughly eight percent of cows calved. Thus there is fairly good evidence that as production has increased over the past two decades, the incidence of retained placenta has not changed.

Mastitis

There are two dimensions to the question of whether the rate of mastitis infection has shifted over time as milk production has increased. The first dimension is the prevalence of subclinical mastitis (measured by average level of somatic cells [SCC]), and second is the incidence of clinical mastitis.

Somatic cell count

Figure 4 shows the herd average log somatic cell count for Minnesota dairies over the past decade.[16] There has been a distinct rise in average cell count for Minnesota dairies over that time. Figure 5 shows the average percent of cows in DHIA herds that were SCC positive (log SCC of 4 or greater) over a two-decade period.[16] The data fluctuate some, but there is a distinct upward trend that a greater proportion of cows are above the traditional cutoff for mastitis. Some of the observed change in these DHIA data might be explained by changes in the population of herds testing with DHIA. If the use of SCC testing gradually extended down to herds with relatively poor management, then the total DHIA average would worsen even if no individual herd performed more poorly. This confounder could be avoided by following herds over time. Ott[18] recently conducted a survey of herds in four regions (Upper Midwest, Mideast, Central and Southwest). In the survey of more than 15,000 herds followed from 1997 to 2001, the average SCC increased from 307,000 to 320,000 cells/ml. Despite efforts to improve milk quality, average levels of subclinical infection seem to be increasing.

Clinical Mastitis

Again, we need to understand the distinction between a biological cause for any apparent association and a management cause. One could hypothesize plausible biological reasons why clinical mastitis might be associated with higher production. High milk production likely causes higher flow rates during milking. Older milking equipment might be poorly adjusted to handle these flows, and the resulting lower teat-end vacuums. Fall-offs and slower milking might be more common in higher producing cows, possibly leading to teat-end lesions and more sub-clinical and clinical mastitis cases. High-producing cows might genetically have more open teats, putting them at greater risk of mastitis. On the other hand, in management terms, a low-producing cow with clinical mastitis is far more likely to be culled than a high-producing cow with the same disease. When surveys are done, low-producing cows have left the herd and may not be included, making it appear that mastitis has been caused by high milk production. If an association is found between high production and mastitis, one still does not know which type of cause is responsible.

Surveying the incidence of clinical mastitis suffers from all of the usual problems of case definition, testing strategy and reporting errors. Published incidence rates vary widely. Canadian data from the late 1970s and early 1980s reported an incidence of 16.8%[3] in the studied herds. State NAHMS data reported 40%[15] and

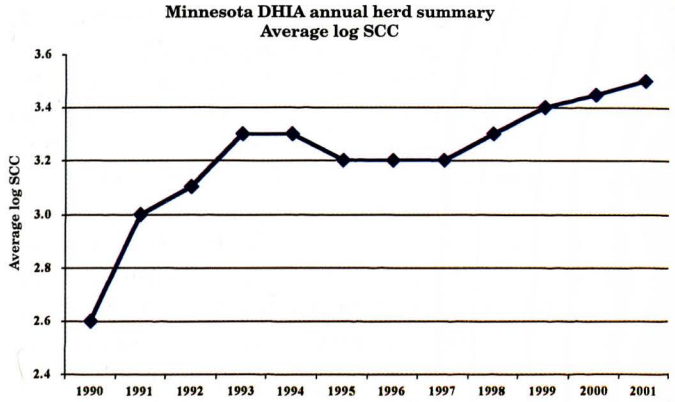

Figure 4. Minnesota DHIA data: average log somatic cell count.

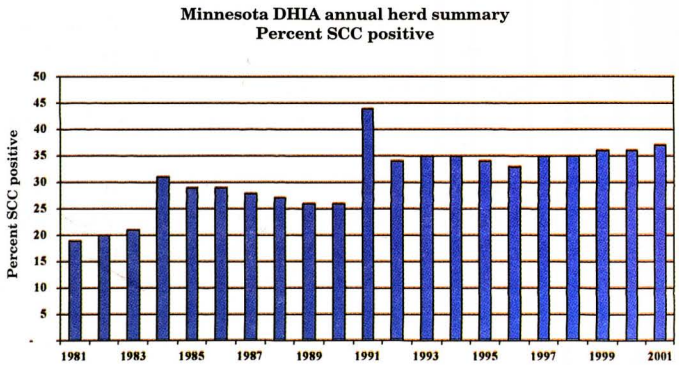

Figure 5. Minnesota DHIA data: percent of the herd with log SCC of 4 or above (SCC positive).

30.3%,[11] while the 1996 national NAHMS survey reported an incidence of 13.4%,[21] and two New York studies reported 9.7%[9] and 14.5%.[10] It is very uncertain whether the incidence of clinical mastitis has changed as production per cow in the industry has increased.

Milk Fever

Rare in first-calf heifers, hypocalcemia, or milk fever, has long been assumed to be associated with milk production. The 1916 USDA *Diseases of Cattle*[20] says: "It is the disease of cows that have been improved in the direction of early maturity, power of rapid fattening, or a heavy yield of milk, and hence it is characteristic of those having great appetites and extraordinary power of digestion." Published incidence rates show a variety of levels. Since the disease is readily diagnosed, variations in incidence probably reflect feeding management in the herds studied more than anything else. In the 1970 veterinary textbook *Bovine Medicine and Surgery*, Kronfeld and Ramberg described the incidence as 3 to 7% of calvings.[13] Canadian data reported an incidence of 2.9% as down cows, and an additional 7.9% as standing milk fevers.[3] A California NAHMS survey reported 4.7%,[11] New York data reported 1.6%[9] and 0.9%,[10] and national NAHMS reported 5.9%.[21] There is little compelling data to suggest a significant increase in the incidence of milk fever as production has increased.

Ketosis

Ketosis is a disease particularly fraught with problems of case identification. The tests are inconsistently applied, and vary in their sensitivity and specificity. Recording and reporting the disease is erratic as well. Kronfeld and Emery described an incidence of 2 to 20% of calvings in their chapter in a 1970 textbook.[14] In the Canadian survey, an incidence of 7.4% was reported,[3] and in the two New York studies an incidence of 4.6%[9] and 5.0%[10] was reported. Again, there is little compelling incidence data (however unreliable) to suggest a significant increase in the incidence of ketosis on dairies as milk production has increased.

General Synopsis of Studies

Using the data detailed above, it seems supportable to conclude that as average production per cow in the dairy industry has increased, over the longer term there has probably been a decrease in reproductive efficiency and an increase in mastitis (at least subclinical mastitis). Since the 1950s, left displacement of the abomasum has become a prominent disease of the digestive tract, but it is more problematic to determine whether the disease has become more common in recent years than it was, for example, in the 1970s. While there are significant concerns about the quality and comparability of data from studies across time and between herds, there is less compelling data that the incidence of ketosis, milk fever, metritis, or retained placenta has increased in the past two decades as production has climbed.

In 1987, Dr. Hollis Erb did a thorough epidemiologic review of available literature on this topic.[7] The summary from that paper states: "Epidemiologic 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'." Dr. Erb's conclusion is supported in general by studies reported after her review.

Cohort Studies

Rather than just looking for associations between production and disease or changes in incidence over time as production has increased, there is an alternative and stronger study approach to the question of production and disease. In this approach, a cohort of cows with known levels of production can be identified at the beginning of the study period and then followed over time, recording cases of clinical disease. By analyzing disease incidence at different levels of production, a more direct measure of the association of production on disease is possible.

In one such study, 2,875 lactation records from 32 commercial herds in Ontario from 1979 to 1981 were considered. Production levels were set by previous lactation Breed Class Average to adjust for differences in ages of cows, and further adjusted for the herd of origin's level of production to remove the confounding effect of the herd's level of production and management.[4] The authors concluded that "the level of milk production was not related significantly to the risk of any of the common disease conditions except for an association between previous production and milk fever." (i.e. no effect on dystocia, retained placenta, displaced abomasum, ketosis, metritis and clinical mastitis, among others). The "study found no association between previous milk production and the risk of reproductive diseases." In summarizing other previous studies of the effect of production on reproduction, the authors concluded "most studies do not indicate major associations between previous production and reproductive diseases and either no association, or a small negative association between

early lactation milk production and reproductive performance."

In a study of 15,320 Holstein cows in 26 herds in New York between 1990 and 1993, associations between milk yield, days open and days to first breeding were investigated.[6] In this study, milk production in the first 60 days of lactation was used as the measure of production and results were adjusted for season, herd and parity. Cows in the highest 20% of production had a slightly lower conception rate than cows in the lowest 20% of production. The authors concluded that "these results indicate that conception and insemination might be influenced by factors related to management (e.g. culling) and to the cow (e.g. disease history), but that increased milk yield plays a very minor role."

In a separate study of 8,070 cows in 25 New York dairies between 1990 and 1993, 305-day milk yield in the previous lactation was studied in association with disease incidence.[9] Higher milk production was not associated with increases in the risk of retained placenta, metritis, ovarian cyst, milk fever, ketosis, or abomasal displacement. There was a small association between increased milk production and clinical mastitis. In this study the authors did not find the small effect on the risk of milk fever found in the Canadian study, but did find an association between production and clinical mastitis not found in the Canadian study.

It may clarify the importance of the measured impact of production on clinical mastitis by quantifying the effect measured in the New York study. Based on the results of the study, increasing production from about 22,300 to about 24,700 lb (10,136 to about 11,227 kg) per lactation (an increase of 2,400 lb [1091 kg] of milk) increased the risk of clinical mastitis 15% (increased the baseline risk of clinical mastitis by a factor of 1.15). If the baseline risk of clinical mastitis at the lower production level was 20% of the herd per year, then the additional 2,400 lb of milk per cow would increase the risk of clinical mastitis to 23% of the herd per year, an increased risk of 3%. Thus if a 100-cow herd increased production by this amount in every cow, they could expect to see approximately three additional cases of mastitis per year.

Disease Effects of Increasing Production Using BST

With the approval of recombinant bovine somatotropin[a] (rBST) use in dairy cows in the US has come another "natural experiment" that can be used to consider whether increased production increases disease. For many diseases, rBST and the subsequent increased milk production cannot be a risk factor because the disease occurs in early lactation, before rBST is administered. Thus the increased production from rBST use cannot increase the risk of retained placenta, ketosis, milk fever, etc., unless a carry-over effect from the previous lactation exists. No such negative carry-over effects of rBST use have been demonstrated. Interestingly, use of rBST does seem to reduce the incidence of ketosis in cows at the start of the subsequent lactation.[5]

For other health problems occurring during the time of administration of rBST, extensive pre-approval and post-approval field studies have shown some negative health impact from the use of rBST and the subsequent increased milk production. The Post-Approval Monitoring Program (PAMP) studied 1,213 cows in 28 herds across the US.[2] In a Canadian government review of rBST for use in Canada, a team of veterinary scientists developed a summary of all available data on the impact of rBST on health.[5] The review included as many as 18 separate studies, depending on the parameter being considered. The review included not only data for the currently marketed form of rBST,[a] but for other experimental formulations of rBST as well. A summary of conclusions from these studies follows:

Reproduction

The Canadian review concluded that rBST use increased average days open by about five days and tended to increase the incidence of cystic ovaries. The review concluded that rBST use resulted in a decreased percent of cows becoming pregnant. There was no conclusive evidence that rBST use increased rates of twinning or abortion.[5] Post-approval studies in the US have found that rBST-supplemented cows are at no increased risk of twinning, have no difference in gestation length, successful calving rate, or percent pregnant, and have not identified a statistically significant increase in days open.[2] The data on reproduction impact is variable and contradictory, but if there is an impact of milk production on reproduction, it seems likely to be small compared to the range of variability in reproductive performance between farms.

Mastitis

The Canadian panel concluded: "Use of rBST increased the risk of clinical mastitis by approximately 25%."[5] It is interesting to compare this estimate of the impact of rBST (and production increases) to the New York study referenced above that showed a 15% increase in the rate of clinical mastitis in cows producing 2,400 lb more milk. If rBST was used from day 60 to day 300 in lactation and production increased 10 lb (4.5 kg) of milk per day, then the total production increase would be the same 2,400 lb. It appears that the magnitude of increased risk of mastitis with increased production, whether from rBST or by other means, is the same order of magnitude. Using the same 100-cow herd example above and a 20% baseline incidence of clinical

mastitis, if the rate increased by 25%, then the new clinical mastitis rate would rise to 25% of cows, or five additional cases per year (.20 * 1.25 = .25). Differing from the Canadian summary of earlier studies, the PAMP study concluded that "Supplementation of cows with rBST had no effect on total mastitis cases, total days of mastitis, duration of mastitis, or the odds ratio of a cow to develop mastitis".[2] Viewed broadly, the data from rBST studies and summaries would seem to indicate that increased production from rBST may put cows at higher risk of clinical mastitis, but at the same level as the effect of any increase in production.

Other health effects

Cows supplemented with rBST have a small increase in the amount of musculoskeletal illness, including foot disorders and disorders of the joints, particularly the hock. The FDA-approved product label reads: "Studies indicated that cows injected with POSILAC had increased numbers of enlarged hocks and lesions (e.g. lacerations, enlargements, calluses) of the knee (carpal region), and that second lactation or older cows had more disorders of the foot region. However, results of these studies did not indicate that use of POSILAC increased lameness."

Conclusion

This paper has looked through three different windows at the question of whether increased production puts a cow at greater risk of disease: 1) changes in rates of disease across the decades as production increased, 2) cohort studies, and 3) the impact of increased production from rBST use. While in some cases it is certain that the risk of illness has increased across the industry as whole (e.g. LDA), it is much more problematic to conclude that the incidence of many of the more common diseases has actually increased in recent decades. It is clear that the dairy industry of today is not the same as the industry of 1950, but at the same time there is little evidence that the industry is suffering from an epidemic of disease as a result of consistent gains in production per cow. Results of cohort studies reach similar conclusions. With the exception of clinical mastitis, there is little evidence that higher producing cows are at risk for increased disease. Finally, rBST and its increased milk production have little impact on cow health beyond clinical mastitis, and minor and variable effects on reproduction and locomotor disease.

In sum, the paradox seems to be largely one of perception, not reality. It is true that healthy, comfortable, well-fed cows make more milk. The technology of dairy farming and the genetics of the cow have changed dramatically in the past decades. Production per cow has increased steadily. In some cases, rates of diseases suffered by dairy cows have changed as well when viewed across the industry as a whole. What has happened at an industry level translates very poorly, if at all, to the individual cow. With a few exceptions, there is little reason to believe that improving production in an individual cow by improving the herd's management will increase her risk of health problems.

The paradox probably arises from the distinction between what happens in the aggregate as the dairy industry's technology has changed, and how those changes will affect an individual cow, such as feeding, housing, genetics, health management, etc. These technology changes may have created the necessary conditions to increase the risk of specific diseases. The risks increase across herds that adopt these technologies. The risk is not necessarily greater for high-producing cows within a herd; in fact, there is at least an argument that these cows are healthier and therefore able to be high producers.

Footnotes

[a]POSILAC 1 STEP®, Monsanto Company, St. Louis, MO 63167.

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