Feeding Dairy Cows for Longevity

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Take Home Messages

- To increase the longevity of dairy cows the focus needs to be on preventing calving/transition-, mastitis-, reproductive- and lameness-related problems.
- There are a myriad of metabolic and digestive disorders that must be minimized during the transition period through management and feeding in order to prevent early-lactation culls or deaths.
- Transition-cow programs are an on-going challenge for dairy producers and their consultants as many interactions between management and nutrition occur on dairy farms that can influence results. Common sense and cow sense continue to be as important to making these programs work on farms as the research base.
- Dietary Vitamin E and selenium are of major importance for reducing mastitis.
- The primary nutritional factors associated with reproductive performance relate to energy and protein status.
- Laminitis is a multi-factorial disorder that requires detailed emphasis on diet formulation, preparation and delivery, feed bunk management, cow management, and cow comfort to control.
- Supplemental dietary biotin improves hoof health.

Introduction

Losses of cows from herds due to involuntary (non-selective) culling or death, and the need for the replacement of lost cows, represent a significant income loss for dairy producers. The average removal (reported as either culled or died) was 34% in an analysis of MN-DHIA records for 718 Holstein herds over four years of calvings (Godden et al., 2003). In the 2002 NAHMS survey (USDA, 2002), 76% of all culls were reported as involuntary culls. Udder or

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mastitis problems, reproductive problems, and lameness/injury comprised 35%, 35%, and 22% of the involuntary culls, respectively (NAHMS, USDA, 2002). Godden et al. (2003) reported that about 25% of the herd removals occurred between 0 and 60 DIM or were likely associated with calving/transition cow issues, and that about 40% of the herd removals occurred after 240 DIM or were likely associated with reproductive failure. A national dairy herd death loss of 4.8% was reported in the 2002 NAHMS survey (USDA, 2002), but death loss averaged 8.1% (range 3.5% to 16.8%) in an analysis of on-farm computerized records from twenty New York herds representing over 12,000 cows (Stone et al., 2005). In the ten herds that recorded their best estimates of cause of death, calving- or transition-related problems were the primary cause for 28% of the deaths and another 10% of the deaths were attributed to mastitis problems (Stone et al., 2005).

The foregoing discussion emphasizes that to increase the longevity of dairy cows the focus needs to be on preventing calving/transition-, mastitis-, reproductive-, and lameness-related culls or deaths. This paper will cover only the nutrition aspects of these areas. It should be noted, however, that management of the cows and their environment is crucial to achieving success in all of these areas and for improving longevity.

■ Nutrition and Transition-Related Culls

Emerging research and extension guidelines on this topic have been extensively reviewed and discussed in papers published in conference proceedings from around the world for over fifteen years. For a concise summary of this area, refer to the 2001 Dairy NRC (NRC, 2001). Nutrient requirements and diet concentrations needed to meet requirements for far-off and close-up dry cows and fresh cows fed example diets are also provided there (NRC, 2001). Pre-partum intake depression and slow intake ascent postpartum, which are major risk factors in the development of metabolic and digestive disorders, make minimizing the incidence of transition-related problems (dystocia, milk fever, ketosis, fatty liver, metritis, displaced abomasum, and sub-acute rumen acidosis) an on-going challenge for dairy producers and their consultants. Body condition at dry-off/calving, dry-period length, length of time on pre-fresh diets, stocking densities in transition pens, abrupt cow movement between facilities, feed delivery, and forage quality are just a few examples of the many interactions between management and nutrition that can influence the results derived from feeding programs designed for transition cows.

■ Nutrition and Mastitis-Related Culls

Vitamin E (740 IU supplemental and 1060 IU total) during the dry period reduced the incidence of clinical mastitis by 37% mastitis in the subsequent lactation relative to un-supplemented controls (Smith et al., 1984). Selenium (Se) injection at 21-d before calving in cows fed selenium deficient diets during the dry period did not affect the incidence of clinical mastitis in the subsequent lactation, but reduced the duration of clinical symptoms by 46% relative to un-supplemented controls; this reduction was 62% for the group supplemented with both Vitamin E and selenium (Smith et al., 1984). Weiss et al. (1997) supplemented Vitamin E at either 100 or 1000 IU during the full dry period or 1000 IU followed by 4000 IU during the far-off and close-up dry periods in diets with a low content of selenium. Quarter incidence of clinical mastitis was 25%, 16.7%, and 2.6% during the first 7-d of lactation for cows receiving 100 IU, 1000 IU, or 1000/4000 IU Vitamin E, respectively, during the dry period. Current Dairy NRC recommendations are for 1000 and 500 IU supplemental Vitamin E during the dry and lactation periods, respectively (NRC, 2001). The dietary selenium requirement listed in Dairy NRC (NRC 2001) and the FDA regulatory limit is 0.3 ppm (DM basis). Other vitamins and trace minerals that play a role in immune function and thus possibly in mastitis prevention include Vitamin A, Beta-carotene, copper, zinc, iron (excess can interfere with copper and zinc absorption), and manganese (NRC, 2001). Dietary Vitamin A and trace mineral recommendations for dry and lactating dairy cows are provided in the 2001 Dairy NRC (NRC, 2001).

■ Nutrition and Reproductive-Related Culls

Severe negative energy balance, excessive mobilization of body fat, and precipitous loss of body condition in early lactation are associated with poor reproductive performance (Wiltbank et al., 2005). Thus, feeding programs and management practices that promote high energy intake, which is a function of both dietary DM intake and energy density, are critically important especially for transition cows. Apart from the role of fat supplements for increasing diet energy density, specialty fat sources with varying fatty acid profiles aimed at improving reproductive performance are being researched and marketed (Thatcher et al., 2004). Feeding diets too high in protein and (or) too low in rumen-fermentable carbohydrates increases blood- and milk-urea nitrogen and has been associated with poor reproductive performance (Ferguson and Chalupa, 1989). Vitamin E and selenium supplementation during the dry period reduced the incidence of retained placenta, metritis, and cystic ovaries (Harrison et al., 1984), which are disorders that can reduce reproductive performance. Dietary Vitamin A and possibly Beta-carotene and trace minerals play a role in reproductive performance (NRC, 2001). Improved

reproductive performance from diets containing above 0.38% phosphorus (DM basis) is highly unlikely (Wu and Satter, 2000; Tallam et al., 2005).

■ Nutrition and Lameness-Related Culls

Readers are referred to several reviews and updates on sub-acute rumen acidosis (SARA) and laminitis (Nocek, 1997; Shaver, 2000; Shaver, 2002; Cook et al., 2004; Nordlund et al., 2004; Stone, 2004; Shaver, 2005). SARA is a prevalent problem for dairy herds (Cook et al., 2004; Nordlund et al., 2004) as characterized by having more than 25% of cows sampled via rumenocentesis 4 to 8 hours after a TMR meal with ruminal pH less than 5.5 (Nordlund et al., 2004). Ruminal pH is largely a function of the balance between the production of volatile fatty acids (VFA) from the fermentation of carbohydrates, their neutralization by salivary and dietary buffers, and their removal by absorption across the rumen wall or passage from the rumen. SARA is caused by the consumption of high amounts of ruminally-available carbohydrate, low amounts of effective fiber, or both (Nocek, 1997). Laminitis, an aseptic inflammation of the dermal layers inside the hoof and a major source of lameness for dairy herds, has been linked to SARA (Nocek, 1997; Cook et al., 2004; Nordlund et al., 2004; Stone, 2004).

Despite diet formulation guidelines for neutral detergent fiber (NDF) from forage, total NDF and non-fiber carbohydrate (NFC; NRC, 2001; Stone, 2004), physically-effective NDF (peNDF; Mertens, 1997; Stone, 2004), and starch (Nocek, 1997) along with the feeding of TMR's, some degree of SARA may be inevitable in high-producing dairy herds because chewing time, and as a consequence salivary buffer flow, decline per unit of rumen-fermentable organic matter (RFOM) intake or VFA production as RFOM intake increases (Shaver, 2005). An increased intake of RFOM as milk production increases is a normal consequence of high milk production, because of increases in DM intake and the feeding of higher-concentrate diets to increase dietary energy density (NRC, 2001). Further, bunk management and cow comfort have been implicated as risk factors for SARA and laminitis in dairy herds (Shaver, 2002; Cook et al., 2004; Stone, 2004). The foregoing discussion highlights the challenges that we face in our efforts to minimize SARA and laminitis in today's dairy herds, and underscores that the margin for error in our feeding programs is small.

Ruminal pH declines following meals with the rate of pH decline increasing as meal size increases and as dietary NDF content decreases (Allen, 1997). Dietary supplementation of sodium bicarbonate attenuates the decline in ruminal pH that is observed post feeding (Erdman, 1988), and may attenuate SARA. The recommended inclusion rate for sodium bicarbonate is 0.75 to 1.0% of TMR dry matter. Keunen et al. (2003) reported that cows with

experimentally-induced SARA did not attenuate SARA by consuming free-choice sodium bicarbonate.

Researchers reported that 20 mg/cow/day supplemental dietary biotin reduced laminitis-related hoof lesions, white-line separation (Hedges et al., 2001; Midla et al., 1998; Potzsch et al., 2003) and sole ulcers (Bergsten et al., 2003), and improved sole ulcer healing (Lischer et al., 2002). Dietary biotin supplementation did not influence ruminal VFA (Zimmerly and Weiss, 2001) or apparent total tract organic matter digestibility (Majee et al., 2003), and it is unlikely that reductions in laminitis-related hoof lesions occur via an attenuation of SARA but rather via biotin's role in keratization of hoof epidermis (Tomlinson et al., 2004).

Nocek et al. (2000) reported reduced laminitis-related hoof lesions in five commercial dairy herds during the year of dietary supplementation with complexed trace minerals (zinc, manganese, copper and cobalt) relative to the year prior when these herds were not supplemented with this complexed trace mineral mixture. The role of trace minerals in keratization of hoof epidermis (Tomlinson et al., 2004) could explain this response.

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