Effects of Low Feed Intake on Gastrointestinal Function[1]

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Introduction

Ration formulation and evaluation systems have advanced significantly over the past 15 yr and how provide a more accurate and detailed prediction for the performance of beef and dairy cattle. Using these models, it is clear that to promote milk and milk component yield for dairy cattle and gain:feed (**G:F**), highly fermentable diets are needed. Feeding highly fermentable diets increases short-chain fatty acid (**SCFA**) production in the rumen and microbial protein over diets with a lower fermentability. Thus, this strategy results in a greater supply of metabolizable energy and increases metabolizable protein supply. On the other hand, feeding rapidly fermentable diets can result in excessive fermentation rates that lead to low ruminal pH, high rumen osmolality, and can increase the concentration of antigenic compounds within the rumen. Obviously, there is a fine balance between diets that promote rapid fermentation and those that cause deleterious effects.

As feeding strategies for ruminants are implemented to promote SCFA production and metabolizable protein, understanding factors that alter the ability of the gastrointestinal tract to maintain its critical functions (**GIT**) is essential. Historically, the role of the GIT was thought to be limited to facilitating digestion, nutrient absorption, and the transit of feed through the GIT. That said, it has been recognized that the GIT plays a critical role in regard to preventing or limiting the passage of non-desired molecules (e.g. microbial associated molecular patterns, pathogenic microbes, and other byproducts arising form fermentation [histamine]) from entering portal circulation. This process is referred to as selective permeability and is one critical component of GIT barrier function. Failure to prevent these molecules from entering portal circulation would result in a systemic immune response. In addition to absorptive and barrier function processes, the gastrointestinal tract also serves as a communicative organ. Recent studies have demonstrated that the GIT can detect nutrients in the lumen that leads to an up regulation of absorptive processes. Moreover, endocrine and paracrine secretions can facilitate communication with the rest of the body including the nervous system. The GIT also communicates with its microbial inhabitants. While much of this research has been conducted in monogastrics, Weimer et al. (2010) has reported that ruminants may also regulate the microbial community structure of the rumen. In that study, they surveyed ruminally cannulated dairy cattle fed the same diet, with similar days in milk, and housed in the same facility. They selected 2 cattle that had the greatest differences in ruminal pH, ruminal SCFA concentrations, and microbial community structure. Subsequently, they completely evacuated the ruminal contents and refilled the rumen with the other cows' contents. Over the next 65 d, they evaluated the microbial community structure and found

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that the microbial profile returned to a profile that resembled the native community. This study provided new evidence that cattle have the ability to at least partially regulate the microbial community structure.

Relevance of Low Feed Intake in Commercial Settings

Beef and dairy cattle are often, albeit inadvertently, exposed to periods of low feed intake (**LFI**). An example for a transient period of low feed intake is for dairy calves during the weaning transition where the increase in starter intake may not compensate for the reduction in milk or milk replacer DMI (Wood et al., 2015). Beef calves also likely reduce DMI in association with weaning as calf walking activity and mealtime is markedly reduced (Price et al, 2003; Haley et al., 2005) although weaning methods can have a significant impact on the negative behavioural response (Haley et al., 2005; Wiese et al., 2015). However, no studies have measured calf DMI prior and post weaning, and only 1 study has attempted to measure calf DMI post-weaning and reported no differences among weaning methods (Wiese et al., 2015). Nevertheless, newly received calves certainly experience extended periods of low feed intake. For example, Hutcheson and Cole (1986) reported that calves in the first, second, and third week after arrival at a feedlot often only consume DM at a rate of 0.5 to 1.5, 1.5 to 2.5%, and 2.5 to 3.5% of BW, respectively. Thus, newly received cattle experience an extended duration of low feed intake and variable extents for the magnitude of intake depression (Hutcheson and Cole, 1986; Loerch and Fluharty, 1999). The LFI occurring for newly received cattle is likely related to the complete feed and water deprivation that occurs during transportation (González et al., 2012) and as part of the response to the change in social structure, physical housing methods, and diets that occur upon arrival.

Transition dairy cattle also experience a transient period of LFI around calving. In fact, a literature review reported that the severity of FR ranges from a reduction of intake up to 68% on d 1 pre-partum relative to 21 d pre-partum for dairy cattle (Hayirli et al., 2002). Although the range in low feed intake is large, on average, cattle reduce their feed intake by 33% with nearly 90% of that reduction occurring in the last week prior to calving. More recent studies have supported the results of Hayirli et al. (2003) as Janovick and Drackley (2010) demonstrated that transition dairy cattle reduced intake by up to 30% as parturition approached and Penner et al., (2007) reported greater than a 30% reduction in primiparous heifers. The extent of LFI can be exacerbated for transition cows in association with infectious diseases or metabolic and digestive disorders (e.g. displaced abomasum, ketosis; Van Winden et al., 2003; Goldhawk et al., 2009) and numerous studies have been able to identify associations between risk for metabolic disease and low feed intake pre-partum (Huzzy et al., 2009). Thus, it is clear that dairy cattle experience a period of low feed intake prior to calving and this is coupled with a rapid increase in DMI and diet fermentability following calving.

Environmental conditions can also induce LFI. Heat stress has been reported to lead to marked reductions in DMI (Maust et al., 1972; Knapp and Grummer, 1991; Holter et al., 1996). The magnitude of reduction in DMI differs based on the severity of the heat stress but under experimental conditions, Baumgard et al. (2011) reported a reduction in

DMI of 18% when exposed to a temperature-humidity index of 64 and Wheelock et al. (2010) reported a DMI reduction of 30% when exposed to the same temperature-humiditiy index as Baumgard et al. (2011). It appears that the magnitude of DMI depression varies among cattle when exposed to the same severity of heat stress and it is likely that the depression in intake is exacerbated with greater severity of the thermal heat load.

Effects of Low Feed Intake on Function of the Gastrointestinal Tract

Although LFI may be transient, past studies in sheep have demonstrated that periods of complete feed deprivation can have negative consequences on the absorptive and barrier functions of the rumen epithelium. For example, the transport of Na^+, Cl^-, Mg^{2+}, and SCFA were reduced by approximately 50% for sheep exposed to a 48-h period of feed deprivation (Gäbel et al., 1993). With respect to barrier function, Gäbel and Aschenbach (2002) demonstrated that the passive passage of a small hydrophilic molecule (3-O-methyl-*α*-D-glucose) was increased following feed deprivation in sheep. While the previous studies provided evidence to suggest that LFI compromises GIT absorptive and barrier function, the experimental model evaluated 48 h of complete feed deprivation; a condition that is not common under industry practices. To address this limitation, we initiated a series of experiments to assess what severities of LFI induce changes in the function of the GIT and to evaluate how the GIT recovers in response to LFI.

In the first study (Zhang et al., 2013a), we assessed the effect of differing severities of LFI by restricting cattle to 75, 50, or 25% of their ad libitum DMI for a 5-d duration. Following exposure to LFI, cattle were provided feed, offered as a total mixed ration, ad libitum and monitored for 3 consecutive weeks. The diet (30% barley silage, 30% grass hay, 32% rolled barley grain, and 8% of a pelleted barley supplement containing minerals and vitamins) was common among all cattle and periods such that we only evaluate the effect of LFI. Our results showed that in response to LFI, the concentration of SCFA in the rumen decreased in a dose-dependent fashion as heifers restricted to 75% had concentrations that were less than when fed ad libitum, concentration of SCFA for heifers restricted to 50% were less than 75%, and those restricted to 25% had concentrations less than those restricted to 50% of their ad libitum DMI. The reduction in SCFA concentrations is logical as DMI decreased, but warrants mention as SCFA provide the bulk majority of the metabolizable energy supply for ruminants and provision of fermentable carbohydrate stimulates the metabolizable protein supply. Corresponding to the reduction in DMI, there was a dose-dependent increase in mean ruminal pH with greatest increases occurring with the most severe LFI. The consequence of the LFI resulted in a tendency for reduced SCFA absorption across the reticulo-rumen and the rate of absorption tended to be reduced as the severity of the LFI increased. Moreover, permeability of the GIT was increased for heifers exposed to 25% of their ad libitum intake. A second study (Albornoz et al., 2013a,b) also found that exposure to LFI (25% of ad libitum DMI) reduced SCFA concentration in the rumen, increased ruminal pH, and reduced SCFA absorption across the reticulo-rumen. It should be recognized that not only did exposure to LFI reduce dietary nutrient supply, but capacity for nutrient absorption was also compromised and permeability was increased. This suggests that exposure to

low feed intake, regardless of the cause (i.e. weaning, heat stress, parturition, transportation, etc.), is likely to reduce metabolizable energy and protein absorption and may predispose cattle to systemic inflammation.

While our research demonstrated that LFI reduces aspects of GIT function, there was no indication for the recovery time required. To address this limitation Zhang et al., (2013b) evaluated the recovery response upon return to full feed allocation. A particularly interesting finding was that the severity of LFI alone caused variability in how rapid heifers returned to ad libitum DMI (Figure 1). For example, heifers exposed to LFI at 25% of their voluntary intake required 3 wk to return to pre-LFI DMI, while those restricted to 75% of their voluntary intake only required 1 wk. This suggests that in the absence of other challenges (parturition, weaning, heat stress, etc.), the severity of LFI can affect the recovery rate for DMI.

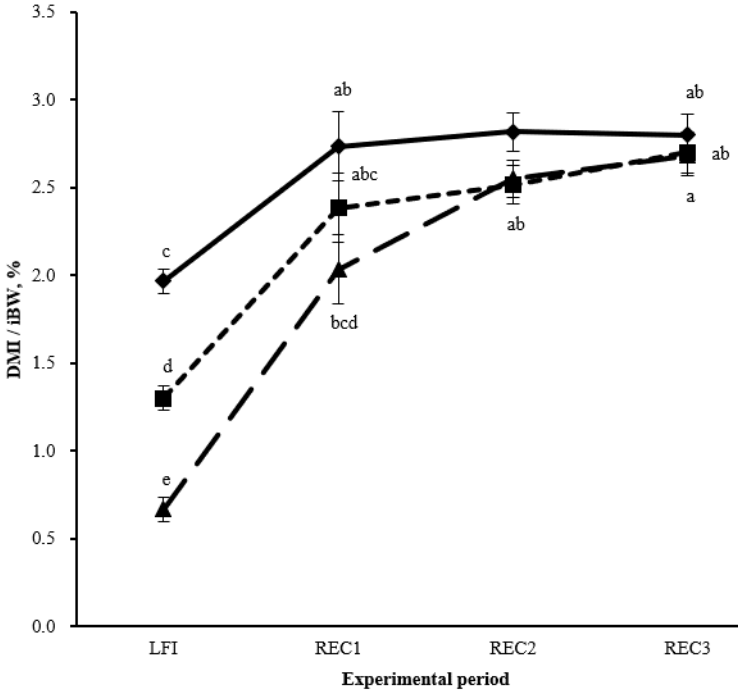

Figure 1. Voluntary recovery of dry matter intake during a 3-wk recovery period (REC1 = 1st week of recovery, REC2 = 2nd week of recovery, and REC3 = 3rd week of recovery) after a 5-d exposure to low feed intake (LFI). For the LFI, heifers were exposed to 75% (solid line with diamonds), 50% (short-dashed line with squares), or 25% (long dashed line with triangles) of their voluntary intake. The DMI was 2.7% of BW prior to LFI. There was a treatment × period interaction ($P < 0.001$) and letters indicate means that differ ($P < 0.05$). Adapted from Zhang et al., (2013b).

Another interesting finding was that despite heifers being fed a diet consisting of 60% forage, return to ad libitum intake resulted in induction of ruminal acidosis and this response was dose-dependent. Thus, heifers exposed to the greatest severity of LFI (25% of ad libitum intake) had the lowest mean ruminal pH (5.88) in the first week of recovery and those restricted to 75% had the greatest mean ruminal pH; although the mean was still only 6.16. On average, the duration that pH was less than 5.5 was 6 h/d during the first week of recovery indicating substantial exposure to low pH conditions. The severity of LFI also affected SCFA absorption during recovery with heifers restricted to 50 and 25% requiring more time to recovery than those restricted to 75%, and permeability of the GIT did not recover for heifers restricted to 25% of their voluntary intake. This data suggests that cattle may require up to 3 wk for the GIT function to recovery after exposure to low feed intake depending on the severity of the LFI event. Given that a greater severity of LFI required more time for recovery, transition dairy cattle that experience metabolic or infectious diseases or feedlot cattle experiencing infectious disease will likely be affected to a greater extent. Moreover, as cattle start to return to their ad libitum intake, they are susceptible to ruminal acidosis, even with low initial feed intake and diets with a substantial forage content.

The data above describe effects of low feed intake on GIT function under artificial conditions and industry relevance could be questioned. To address this issue, we evaluated the changes in DMI and total tract permeability of the GIT for dairy calves at weaning (Wood et al., 2015). In this study, 14 Holstein bull calves were used and fed milk replacer at a rate of 15% of their BW (adjusted weekly). The milk replacer was included at 150 g (DM basis)/1 L of water. Calves were exposed to a 42-d milk feeding protocol followed by a 7-d step down weaning program (**WEAN**) or were not weaned (**CON**). The step-down weaning protocol was effective at increasing starter intake as WEAN calves had greater intake than the CON. Calves in the WEAN treatment experienced did not increase BW to the same extend during the step-down period further indicating a reduction in nutrient intake associated with weaning. To evaluate GIT permeability, we orally dosed Cr-EDTA and measured its appearance in urine. This approach is the same method used and validated by Zhang et al. (2013). We observed that permeability of the GIT decreased from wk 2 of age to wk 4 of age and for the CON continued to decrease to wk 6. However, calves that were weaned followed the same trend until wk 6 where weaning caused a marked increase in permeability of the GIT. This supported the work of Zhang et al. (2013a,b) showing that reductions in DMI and nutrient intake are associated with increased GIT permeability.

Strategies to Minimize the Negative Effect of Low Feed Intake and Accelerate Recovery of the Gastrointestinal Tract

Based on the data above, it appears that LFI consistently decreases aspects of GIT function. However, this data does not indicate what management strategies can be used to mitigate the negative effect caused by LFI or accelerate the recovery response. It should be recognized that there are instances where LFI could be predicted (i.e. weaning, transportation) and hence dietary scenarios could be put in place to minimize the negative effect of LFI. Alternatively, there are many scenarios where LFI cannot be

accurately predicted (metabolic disorders and infectious disease) or the timeline may be difficult to accurately predict (parturition and heat stress). Thus, there is a need to evaluate strategies that can either mitigate the effect of LFI when it is predictable and, more practically, accelerate the recovery following a period of LFI.

For predictable exposure to LFI, we evaluate whether altering the forage:concentrate ratio of the diet could be a mechanism to mitigate the negative effect (Albornoz et al., 2013a,b). In this study heifers were either fed a high forage diet (92% forage with 50% of the forage supplied from barley silage and 50% from grass hay, and 8% of a pelleted barley supplement containing minerals and vitamins; DM basis) or a moderate forage diet (30% barley silage, 30% grass hay, 32% rolled barley grain, and 8% of a pelleted barley supplement containing minerals and vitamins; DM basis). These treatments were used as feeding a high-forage diet may increase ruminal retention thus decreasing the severity of the LFI event, while feeding a moderate forage diet provides a greater nutrient supply per unit of feed intake. Heifers were exposed to a 5-d period of LFI with feed restricted to 25% of voluntary intake. As previously reported LFI reduced SCFA in the rumen, increased mean pH, and reduced the rate of SCFA absorption. Feeding the high forage diet prior to and during LFI, increased serum non-esterified fatty acids (an indicator of adipose tissue mobilization) to a greater extent than the moderate forage diet, and reduced the rate of recovery for DMI after return to voluntary intake. Heifers fed the high forage diet prior to and during the LFI also had greater risk for ruminal acidosis during the recovery period. Surprisingly, the diet fed prior to and during LFI did not affect the rate of recovery for SCFA absorption. This indicates that diets with a high forage content may have a more deleterious effect than diets with a moderate forage content when fed during a period of LFI, likely because of reduced nutrient density.

On the other hand, most episodes of LFI will not be accurately predicted but can be detected with adequate feed bunk management and behavioural responses. Given that cattle are susceptible to ruminal acidosis during recovery after LFI, we evaluated the effect of feeding a moderate or high forage diet during recovery. Interestingly, heifers fed the high forage diet following LFI resumed voluntary DMI within the first wk of recovery whereas; heifers fed the moderate forage diet required 3 wk. The response of the moderate forage-fed heifers mirrored that reported by Zhang et al. (2013b). Feeding the high-forage diet during recovery also allowed heifers to stabilize mean pH and those fed the high forage diet did not experience ruminal acidosis. Heifers fed the moderate forage diet had mean pH below 6 during the first week of recover whereas, heifers fed the high forage diet had a mean pH above 6.3. The induction of ruminal acidosis during the first week of recovery was also evident as heifers fed the moderate forage diet spent over 4 h below pH 55 while those on the high forage diet spent less than 20 min below the same threshold. Although ruminal fermentation parameters were affected, there were no differences in the rate of SCFA absorption between recovery treatment strategies.

To apply such findings to the feedlot sector, a study was conducted to evaluate whether feeding a diet lower in barley grain during recovery or feeding a diet lower in barley grain with a cocktail additive could help accelerate recovery of GIT function (Penner et al., unpublished). In this study 32 lambs were assigned to 1 of 4 treatments. The treatments consisted of a finishing ration (9% barley silage, 79% barley grain, and 12% of a barley-based mineral and vitamin supplement) throughout the study (CON) or lambs that were fed the finishing ration but exposed to a 3-d period of LFI at 50% of voluntary intake and then 1 of 3 recovery treatments. The recovery period was 5-d. To evaluate the recovery response after LFI, lambs were either fed the finishing ration (FIN), or 1 of 2 diets where the proportion of barley silage was increased to 20% at the expense of barley grain. This approach is commonly referred to as a 'storm' diet in the feedlot sector. The second 'storm' diet also included a dietary additive of rumen protected betaine (0.7% of DM), superoxide dismutase (0.01% of DM) as an antioxidant, and Na-butyrate (0.2% DM). Betaine has been reported to help support GIT function during coccidia challenges (Kettunen et al., 2001; Fetterer et al., 2003), and superoxide dismutase has been reported to improve GIT function in mice (Vouldoukis et al. 2004). Moreover, there is evidence that the ruminal epithelia may experience hypoxic conditions (Dengler et al., 2015) supporting that antioxidants may have a beneficial role in the ruminant GIT. Finally, butyrate has been shown to induce positive effects a low does (Gorka et al., 2007; Kowalski et al., 2015). We observed that the CON group did not change DMI throughout the study, thereby serving as an appropriate control as they were not exposed to a LFI challenge. Interestingly, lambs fed the STORM or STORM plus additive diets during recovery increased DMI relative to that during LFI, while lambs fed the FIN diet did not increase DMI during recovery. This suggests, that increasing the proportion of forage after a period of LFI can help recovery of DMI when fed finishing diets. While all treatments, except the CON, had lower ruminal pH during recovery than during the LFI challenge, the STORM and STORM plus additive diets had numerically greater ruminal pH during the 5-d recovery than lambs provided the finishing diet. We also found that lambs fed the STORM plus additive diet tended to have greater rates of acetate absorption and had greater butyrate absorption in the recovery period than the other treatments. This study demonstrated that moderate increases in the forage proportion can help cattle recover after a period of LFI, even with finishing diets, and that provision of additives reported to accelerate GIT function can help the recovery response. Future research is needed to evaluate which additives are most beneficial to improve the recovery of the GIT.

Summary

Low feed intake negatively affects absorptive and barrier functions of the gastrointestinal tract and predisposes cattle to ruminal acidosis. Feeding a diet with a greater energy density prior to low feed intake can help accelerate the recovery response as can feeding a diet with a high forage content after a period of low feed intake. In a finishing scenario, this can be accomplished simply by increasing the proportion of forage in the diet (by approximately 11% units). We also report that feeding additives that support gastrointestinal function (e.g. betaine, superoxide dismutase, and butyrate), can help accelerate the recovery response for the gastrointestinal tract. These results demonstrate

an industry-relevant nutritional challenge along with strategies that can be implemented to mitigate the impact of low feed intake.

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