Gut Health During Diet Transitions

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Diet Transitions and Rumen Health

Throughout the life of a ruminant, diets are changed to match the needs of the new state. Going from a dry-cow diet to an early lactation represents one of the extreme diet changes that cows undergo. While the cow may be switched over in a matter of several days, the structural and cellular adaptations that need to happen for the cow to fully adapt to the new diet takes weeks. During the transition period, structural adaptations begin in the first week of lactation, and persist to at least 6 weeks of lactation (Laarman et al., 2015, Steele et al., 2015). Until a cow fully adapts to the new diet, there is an elevated risk of VFA production in the rumen exceeding VFA removal from the rumen. Left unchecked, this excess fermentation can lead to subacute ruminal acidosis.

Subacute ruminal acidosis occurs when rumen pH drops below 5.8 (Aschenbach et al., 2011), and is estimated to cost approximately $2.12 per head per day (Stone, 2004). Losses from subacute ruminal acidosis largely come from decreased milk fat production, which is positively correlated to rumen pH (Allen, 1997). In addition, subacute ruminal acidosis is linked to laminitis, rumenitis, and liver abscesses (Plaizier et al., 2008), which increase cull rate. Improving rumen health during dietary transitions, then, is a function of preventing and mitigating the rumen health challenges caused by subacute ruminal acidosis.

Subacute ruminal acidosis strains the barrier integrity of the rumen epithelium, which prevents ruminal bacteria from entering the cow's bloodstream and causing disease. The rumen epithelium contains 4 layers of cells that form a physical barrier between the rumen microbes and the bloodstream (**Figure 1**). Between cells of the stratum granulosum and stratum spinosum are anchors that physically hold cells

together, called tight junction proteins (Graham and Simmons, 2005). Together, tight junction proteins and the epithelial cells form the physical barrier between the rumen and the bloodstream. Integrity of this barrier is paramount to health of the rumen.

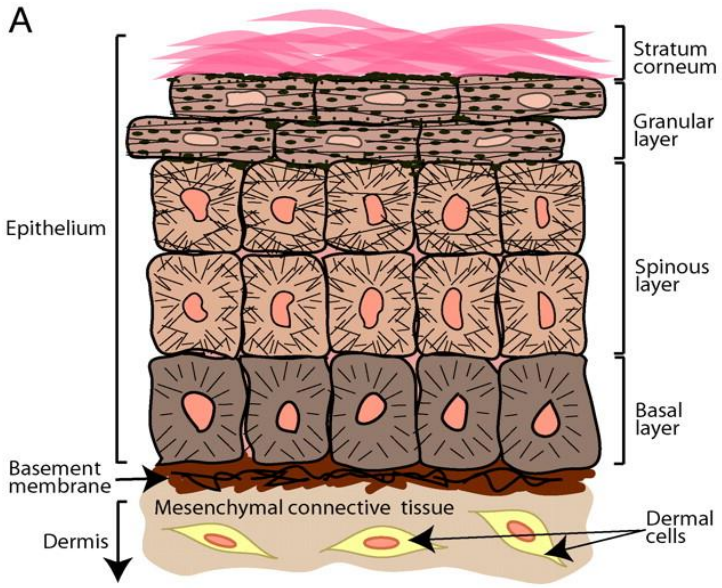

Figure 1. Lining the rumen is a 4-layer epithelium that prevents bacteria from entering the bloodstream, leading to liver abscesses and laminitis (Alonso and Fuchs, 2003; Copyright 2003, National Academy of Science. Used for non-commercial purposes).

The barrier integrity of the rumen epithelium is strained by subacute ruminal acidosis, especially during a dietary change. Steele et al. switched cows over from a high forage diet to a high grain diet over the course of 4 days. The result was extensive break-up of the rumen papillae, exposing layers of the papillae to the rumen that normally are protected by the corneal layer (**Figure 2**; Steele et al., 2011). Detailed microscope images show the disintegration of the epithelium both by the sloughing and by the gaps that begin to open between cells of the epithelium. We can see barrier integrity of the rumen epithelium is seriously challenged by subacute ruminal acidosis following a dietary change. As a result, dietary changes pose a significant challenge to rumen health, and needs to be managed carefully.

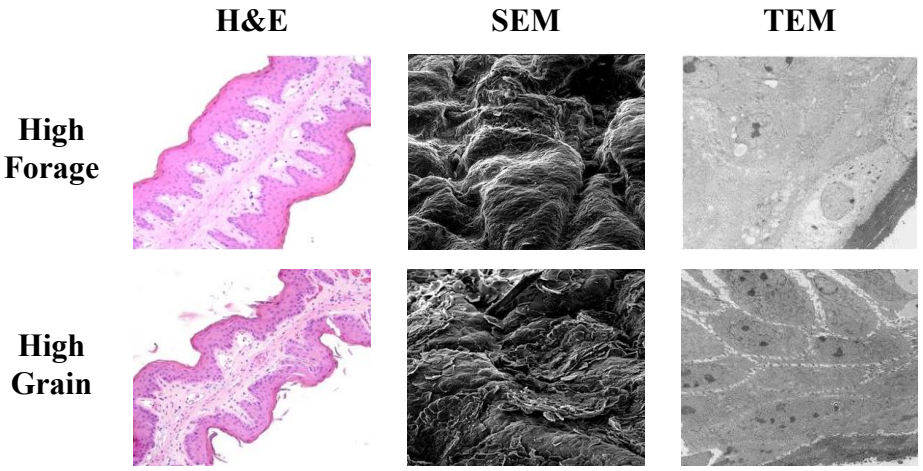

Figure 2. Damage to rumen epithelium caused by subacute ruminal acidosis. Sloughing of the outer layer facing the rumen (stratum corneum) can be seen in the H&E stain and the scanning electron micrograph (SEM). Gaps between the epithelial cells and a failure of barrier integrity is evident in the transmission electron micrograph (TEM; Steele et al., 2011).

Improving Rumen Health by Increasing VFA Uptake

Managing rumen health during dietary transitions relies on increasing VFA uptake. During the transition period, NFC content of the diet rapidly increases to as high as 45% (AlZahal et al., 2014), which depresses rumen pH (Laarman et al., 2015). Depressed rumen pH can be avoided if the rumen adapts to increase VFA uptake. In a study in 2011, we separated pre-weaned dairy calves into 2 groups. The control group received milk and hay only, and the starter group received milk, hay, and calf starter. Despite increased VFA concentration in the calf starter group indicating increased fermentation, rumen pH was not different between the 2 groups (Table 1; Laarman and Oba, 2011). This suggests the rumen adapted to the increased diet fermentability by removing more VFA from the rumen.

Table 1. Average rumen pH of pre-weaned Holstein dairy calves fed 2 different diets (Laarman and Oba, 2011)

	Milk & Hay	Milk & Hay & Starter
Average pH	6.42 ± 0.10	6.27 ± 0.12
Duration pH<5.8, min/d	101 ± 100	237 ± 126
Total VFA, mM	64.6 ± 8.6	99.1 ± 8.1*
Starter DMI, kg/d	N/A	0.76 ± 0.04
Hay DMI, kg/d	0.23 ± 0.07	0.34 ± 0.8

*$P < 0.05$

To avoid subacute ruminal acidosis, either VFA production has to decrease or VFA removal must increase. A previous study demonstrated an increase in absorptive area of the rumen (Dirksen et al., 1985), which increases VFA absorptive capacity in the rumen. Another study, in sheep, showed the ability to remove VFA from the rumen was directly related to resistance to subacute ruminal acidosis. Sheep with a higher rate of VFA removal from the rumen were more resistant to a subacute ruminal acidosis challenge than sheep with a lower rate of VFA removal from the rumen (Penner et al., 2009). Therefore, improving VFA removal from the rumen is key to preventing subacute ruminal acidosis and improving rumen health.

Increasing VFA removal from the rumen requires improving the mechanisms for VFA transport away from the rumen. One primary candidate for this is a transporter embedded in the rumen epithelium called monocarboxylate co-transporter 1 (MCT1). In past studies, this transporter was linked to VFA removal from the cow caecum (Kirat and Kato, 2006) and goat rumen (Kirat et al., 2006). Using dietary means to increasing MCT1 might, then, help improve resistance to subacute ruminal acidosis, and lead to better rumen health.

Improving resistance to subacute ruminal acidosis requires finding ways of increasing MCT1 to improve ruminal VFA removal. In calves fed a typical calf starter, both butyrate and MCT1 increased (Laarman et al., 2012). Part of the explanation may be that butyrate affects a large number of cellular genes, which affects how cells function (Davie, 2003, Kiela et al., 2007). Bringing that knowledge to the farm, we supplied lactating dairy cows with supplemental butyrate. Cows were abruptly switched to a high-grain diet (45% NFC) over the course of 2 days, then were ruminally dosed with butyrate at the rate of 2.5% of their DMI, twice daily for 7 days (Laarman et al., 2013a). From this study, we saw increased butyrate concentrations in the rumen, increased BHBA concentrations in the blood (indicating butyrate metabolism), and increased MCT1 (Laarman et al., 2013a). At the same time, we saw genomic changes suggesting improved barrier integrity (Laarman et al., 2013b), which was corroborated by other studies showing the positive effects of butyrate on barrier integrity (Hamer et al., 2008, Baldwin et al., 2012). These studies suggest butyrate improves both VFA uptake and barrier integrity of the rumen.

In short, the linkage of VFA transport with resistance to subacute ruminal acidosis means rumen health can be improved by strategies that optimize VFA transport and improve barrier integrity. Such strategies confer benefits to ruminants by increasing nutrient absorption, increasing rumen pH, and improving rumen health. Butyrate shows much potential for improving gut health through these various means, but more work remains to be done to effectively use butyrate to improve gut health in ruminants.

Strategies for Optimal Rumen Health

How can we use feeding strategies to improve rumen health on farm? One promising area of promoting rumen health is through increasing ruminal butyrate concentrations, as outlined previously. Because butyrate has been linked to SCFA transport (Kirat and Kato, 2005; Kirat et al., 2006), and improving barrier integrity (Hamer et al., 2008, Laarman et al., 2013b), butyrate holds considerable promise as a gut health promoter. In poultry, trace butyrate inclusion at 700 ppm decreases feed: gain from 1.80 to

1.73 (Chamba et al., 2014), so dietary butyrate increases animal performance. At this point, more research needs to be done on how to effectively include butyrate or butyrate pre-cursors in the ruminant diet.

Lastly, another candidate for improving gut health is probiotic feed additives, which can increase butyrate production in the rumen. In one recent study, two groups of lactating cows were switched from a high forage diet (44.9 % NDF, 31.5% NFC) to a high-grain diet (28.2% NDF, 45.2% NFC) over the course of 2 days. One of the groups was also fed an active dry yeast additive as a top dressing, and cows were allowed to adapt for 21 days. After 21 days, cows fed the active dry yeast had decreased incidence of subacute ruminal acidosis, down from 550 min/d to 130 min/d (AlZahal et al., 2014). Coupled with a decrease in subacute ruminal acidosis was an increase in both DMI and 4% fat corrected milk yield (AlZahal et al., 2014). Through the use of probiotics, we may be able to improve fermentation, and increase butyrate concentrations in the rumen.

Conclusion

From weaning to lactation, dietary changes in ruminants put a tremendous stress on gut health. Gut health during diet transitions is augmented when the rumen adapts to match VFA uptake to VFA production in the rumen. Increased VFA uptake is already linked to increased resistance to subacute ruminal acidosis, so the challenge ahead lies in strategies to improve VFA transport. Increasing ruminal butyrate concentrations, whether through direct addition to the feed, or via probiotic use, contributes to tackling the challenge of diet adaptation in the rumen. Through meeting this challenge, we can make strides in improving rumen health and ruminant productivity.

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