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Mini Review

Physical and Metabolic Constraints on Feed Intake in Ruminants

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Abstract

Feed intake control in ruminants is mediated through physical and metabolic constraints. Rumen fill, dietary fiber concentration, and fiber digestibility are important physical constraints. Rumen Volatile Fatty Acids (VFA) concentrations, post-rumen nutrient assimilation and absorption, and hepatic and systemic nutrient balance (and imbalance) are important metabolic constraints on feed intake. Blood levels of glucose and some ketones and fatty acids are other significant players in feed intake regulation in ruminants. Research is needed to elucidate how to optimize feeding strategies and feeding systems to improve feed intake in high-producing ruminants.

Keywords: Feed Intake; Metabolism; Constraint; Ruminant

Objective and Physical Constraints

The objective of this review article was to elucidate the main physical and metabolic constraints on feed intake in ruminants. Over the last 25 years, several major physical and metabolic regulators of feed intake in ruminants have been emphasized. Ruminal fill [1-4] is one of the central regulators of Dry Matter Intake (DMI) under certain circumstances such as when feeds with low digestibility are fed [5]. The dietary NDF, especially from forage, is a key contributor to reticulorumen fill. The greater NDF lowers the clearance rate of the rumen contents [6]. Hence, the dietary NDF can be a key controller of feed intake in early and peak lactation cows that have not peaked in DMI or with limited rumen fiber pool [2]. The NDF digestibility can significantly impact DMI [7]. As NDF digestibility increases, the depressing effect of NDF on DMI weakens. Allen [2] stated that DMI rose by 0.17kg per unit rise in *in vitro* or in situ NDF digestibility. At higher NDF digestibility, the NDF will have a smaller impact on rumen distension. Thus, factors affecting NDF digestibility will affect DMI.

Metabolic Constraints

Among the important metabolic constraints of appetite are rumen concentrations of volatile fatty acids [8,9]. Propionate injection into the portal vein has reduced feed intake in sheep [10,11]. Propionate rather than acetate seems to cause hypophagia [2]. Insulin secretion [12] and hepatic receptors [10] have been proposed to mediate the hypophagic effects of propionate. In addition to the hepatic chemoreceptors, hepatic thermoreceptors may also control feed intake. Di Bella et al. [13] heated the rat liver artificially and observed an increased chewing activity with reduced feed intake.

Feed intake is ultimately a psychological phenomenon integrating animal's abilities to cope with changes in diet composition and metabolic demands [14]. Thus, one must consider that the rumen or blood VFA is only one of many factors involved in feed intake [15]. Illius and Jessop [16] suggested that imbalances in nutrient supply both in the rumen, postrumen, and hepatic levels can reduce feed intake. They proposed that maximizing acetate use for lipogenesis needs a synchronous glucose supply. Glucose fuels lipogenesis by providing ATP and cofactors such as NADPH needed for fatty acid elongation [16]. Thus, even the high production rate of acetate, if accompanied by adequate supply of other nutrients, May not necessarily down-regulate feed intake. The framework of Illius and Jessop [16] presumes that nutrient imbalances constrain feed intake via accumulation of excess metabolites such as acetate. Therefore, the animal targets a level of intake that minimizes nutrient imbalances. According to this framework, in the absence of adequate glucose, acetate will mount up and act as a hypophagic feedback.

β-hydroxybutyrate (BHBA) is another metabolite that can contribute to feed intake regulation. Subcutaneous administration of BHBA reduces feed intake in rats [17,18]. The satiety signals may arise partly from direct oxidation of BHBA. Consequently, reducing equivalents or NADH accumulate in the mitochondria and depress feed intake [19]. Unlike BHBA, subcutaneous administration of acetoacetate does not affect feed intake [17]. It seems, therefore, that the process of hepatic BHBA conversion to acetoacetate involving other metabolites and co-factors and not acetoacetate per se influences satiety. The role of BHBA on feed intake regulation needs further research in ruminants.

Mayer [20] was the first to suggest that blood glucose controls feed intake. Mayer [20] indicated that the hypothalamus takes up glucose and thereby monitors and controls peripheral blood glucose. According to the Mayer's glucostatic theory, the hypothalamus controls blood glucose by controlling feed intake. Early trials with intra-ruminal, intra-venous, or intra-cerebroventricular glucose infusion in sheep [21], goats [22,23] and cattle [24] demonstrated no effects of glucose on feed intake. Blood glucose and its diurnal fluctuations are considerably lower in ruminants than in non-ruminants [25]. Thus, blood glucose does not seem to be as significant in controlling feed intake in ruminants as it is in non-ruminants. This is not surprising, because due to the extensive rumen fermentation of dietary carbohydrates, VFA and not glucose are the main digestion end-products absorbed across the gut in ruminants [26]. When high-starch diets containing corn and sorghum grains are fed, however, the

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amount of intact or partially hydrolyzed starch escaping the rumen may increase [27]. The intestinal starch and the resulting glucose may affect feed intake. The role of the absorbed glucose across small intestine on feed intake regulation requires has not been elucidated.

Implications

Physical and metabolic constraints on feed intake in ruminants were reviewed. Optimal feeding strategies and feeding systems must be adopted to regulate feed intake such that rumen and intermediary metabolism can be optimized. Research is needed to elucidate how to optimize feeding strategies and feeding systems to improve feed intake in high-producing ruminants.

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