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Practical aspects of urea and ammonia metabolism in ruminants

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Abstract

Nitrogen was recognized over 200 yr ago as an element essential for normal function of farm animals. During the first half of the 19th century, the roles of proteins and urea in N metabolism were discovered. By the middle of the 20th century, the substrates, products, and enzymes of the urea cycle were elucidated. Work since then has quantified dietary crude protein requirements for specific production goals, protein synthesis and breakdown, ruminal ammonia production, endogenous urea synthesis, and urea recycling. In ruminants fed conventional diets, N absorbed as ammonia can be several times the amount of N absorbed in the form of amino acids or peptides. Nitrogen recycled to the digestive tract as urea in saliva or urea transported from blood ranges from 10 to 40% of N consumed in feed. Under production conditions, from 0 to 20% of N consumed by ruminants is retained as tissue N or excreted as milk protein. This review describes the quantitative aspects of urea and ammonia metabolism in ruminants and it relates the metabolic or economic costs of that metabolism to practical feeding situations. The review concludes with a discussion of conflicts and considerations among three main priorities in ruminant N metabolism: 1) maximizing microbial function in the rumen; 2) optimizing amino acid supply to the host ruminant; and 3) minimizing negative environmental effects of cycling N through ruminant production systems.

Key Words: Ruminants, Urea, Ammonia, Nitrogen

Introduction

Our information and practical applications of protein nutrition evolved from identification and characterization of basic organic elements, through characterization of whole animal needs, and recently arrived at the description of cellular and subcellular metabolic processes. The connection of protein to other nonprotein, nitrogenous compounds in ruminant nutrition came with the discovery of the interactions between ruminants and their enteric symbionts. This review focuses on nonprotein aspects of nitrogen metabolism in ruminants. We have reviewed the history and basic aspects of ammonia and urea metabolism and discussed some practical applications of current information. We relied on previous reviews of related topics, particularly those of Meijer et al. (1990), Tamminga (1992), Carpenter (1994), Parker et al. (1995), Van Horn et al. (1996), and Wallace (1996).

Discussion

The History of Nitrogen 1614–1932

Before 1614, nutrition centered around the classic Greek thought that individual temperaments were affected by the foods that were eaten. The first influential research to start the basis of a more scientific approach to the area of nutrition was conducted by an Italian scientist, Santorio, who conducted one of the earliest balance trials upon himself and determined that whereas he consumed approximately 3.6 kg/d, he only excreted approximately 1.4 kg/d. He attributed the unexplained disappearance of about 2.2 kg of material a day to "insensible perspiration" (Carpenter, 1994). In 1663, Boerhaave established the mechanical theory of nutrition that was founded on the idea that the "human body itself seems to be but an engine." This idea was further promoted by Pitcairn in 1718, who declared that animal heat was a result of the friction of particles within vessels. As a result, there would be holes worn in the vessels that would have to be filled by food. This helped to establish why laborers should not consume the daintier foods such as meat, because they would be burned up too quickly to do them any good. Hence, the working force should consume coarser foods that would be able to remain in the system long enough to fill in their vascular "holes" and leave the more easily assimilated meats and such to the less-active upper classes whose bodies could better utilize such foods.

Near the end of the 17th century, however, a new theory was being devised that likened the digestion of food to a fermentation process, with animal products fermenting to produce an alkalotic product that, as Boyle noted in 1684, was not unlike smelling salts (NH_4CO_3), and grains fermenting to form well-observed acidic products. However, this theory was challenged with the obvious question of how herbivores could exist on a strictly acid-forming diet and convert it into alkalotic animal products. This question was countered by the discovery by Beccari in 1745 that wheat contained an "animal substance" (gluten) after washing out the floury particles. This was then thought to explain to some extent how animals could survive on a purely herbivorous diet by using this "animal substance" to repair body tissue and the starchy portions as fuel.

In 1785, Berthollet reported three of his main results on the composition of the volatile alkali associated with animal substances to the Academy of Sciences in Paris: 1) reacting

"volatile alkali" (ammonia gas) with chlorine resulted in the production of N gas; 2) reacting ammonia gas with hot metallic oxides resulted in the production of hydrogen; and 3) electric sparking in ammonia gas gave a greatly increased volume of gas that was then burned in oxygen with the production of water (proving the presence of hydrogen) and left an unreactive gas (assumed to be N) (Carpenter, 1984). Along with Berthollet, others contributed to further defining N and N-containing substances. In particular, in 1789 Lavoisier led studies of nutrition in a new direction, with his Traité élémentaire de chimie, which listed several major elements of living bodies: carbon, hydrogen, oxygen, and N (azote).

In 1813, Magendie established the necessity of N-containing compounds to maintain life in dogs. Dogs were fed diets containing only pure sugar and distilled water or diets in which olive oil, gum, or butter replaced the sugar. All of the experiments, which did not have any positive controls, resulted in the death of the dogs within 40 d. Boussingault in 1836 provided some basic concepts of N and its role in nutrition that are considered valid today. He noted that all the vegetable substances used as food contained N. He also noted that the nutritional quality of a vegetable substance was proportional to its content of "animal substances"; however, some N-containing substances obtained from vegetable products were not nutritive. With these basic concepts in mind, he began to establish a standard of evaluating the nutritive quality of foods based on N content, even though he himself admitted that this was not the sole source of nutritional value to be derived from foods. He was also the first to discover fixation of N by certain plants and that non-N-fixing plants could have increased yields when planted after a N-fixing plant. This supported his theory that while some plants could "fix" atmospheric N, others could not and required the presence of already formed N-containing compounds in the soil. Finally, he also performed the first N balance trials with farm animals. The results of these experiments suggested that animals did not utilize atmospheric N for anabolic purposes, because the diets of the animals he used provided more N than was excreted within the given period.

During the mid 1800s, amid rampant infatuation with the newly described protein radical, Leibig argued that urea production was the result of the autoconsumption of muscle tissues as they expended their force. As such, he further argued that N from food cannot be excreted as urea without first being part of organized tissue. Leibig's theory was later discredited by Bischoff and Voit, his colleague and student.

As the 19th century drew to a close, the role of nonprotein N in ruminant systems was recognized by Zuntz in 1891. He has been credited as the first to suggest the role that rumen microflora play in the utilization of nonprotein N by ruminants. This was in contrast to the popular concept of the era, which was even supported by Zuntz himself, that nonprotein N had no nutritive value to animals and should be removed from feed samples prior to analysis of their available protein.

Although many had speculated on how urea was produced and used in the body, it was not until the work of Krebs in the early 1930s that the current concept was established. After observing that in vitro production rate of urea was increased by the addition of ammonium salts to saline solutions that also contained liver slices, Krebs and Henseleit (1932) discovered how urea is produced by ureotelic animals. After noting that many of the saline mediums of the time were deficient in two major plasma constituents (HCO_3^- and CO_2), Krebs and a medical student, Henseleit, devised a medium that would much more closely mimic physiological plasma (Krebs, 1981). After establishing this modified medium and testing its relevance, they began a systematic measurement of urea production in the presence of a variety of chemical precursors. This led to the discovery that urea was synthesized at exceptionally high rates when both ornithine and ammonium ions were present. While at first uncertain as to whether ornithine was acting as a N donor to urea, it was determined that over 20 additional molecules of urea could be produced from a single ornithine molecule, provided that there was sufficient ammonium present. This led to the concept of ornithine as a catalyst in the formation of urea. With the acknowledgment of the catalytic action of ornithine, it was decided that there must be some intermediate forms that had to be formed prior to the actual production of urea. They recognized that there was probably even more than one intermediate because ornithine, CO_2, and ammonia, interacted to form urea. It was known at this time that there was a correlation between the presence of arginase and urea production as well. They then demonstrated the rapid formation of urea in the presence of citrulline and ammonium: citrulline + NH_4^+ + arginine + $H_2O \rightarrow$ ornithine + urea. These results led to the development of the ornithine cycle (Krebs and Henseleit, 1932) that forms the fulcrum for research involving N flux in animal systems.

Basics of Ammonia Metabolism

Because physiological pH is usually 2 pH units or more less than the ammonia-ammonium pKa, essentially all ammonia in ruminants or their gastrointestinal tract is in the protonated, NH_4^+ form. Protonation and deprotonation of ammonia provides a mechanism for movement of the molecule across membranes, and they make ammonia an important intermediate in acid-base regulation as well as N metabolism. For purposes of this discussion, "ammonia" will include both ammonia and ammonium, unless otherwise specified.

Dietary sources of N include nucleic acids, amino acids, proteins, peptides, amines, amides, nitrates, nitrites, urea, and ammonia. Endogenous sources include sloughed cells and urea that reenters the rumen across the ruminal epithelium or in saliva. With the exception of some proteins and N associated with ADF, these N sources are readily soluble and susceptible to degradation in the rumen. Evolution of symbiosis among ruminal microbes and their host, as well as symbiosis among the microbes themselves, has

placed ammonia as a major component of N metabolism in ruminants. Many of the cellulolytic bacteria prefer or require N in the form of ammonia (Russell et al., 1992), thus forming a link between transformation of N sources listed above to ammonia and fermentation of fiber. In vitro estimates of the proportion of microbial protein derived from ammonia with a variety of substrate (dietary) protein sources ranged from 40 to 68% (Hristov and Broderick, 1994). Wallace (1996) divided ammonia-producing bacteria into those in high numbers ($> 1 \times 10^9$/mL ruminal fluid) with low activity (10 to 20 mmol $NH_3 \cdot min^{-1} \cdot mg^{-1}$ protein; *B. fibrisolvens, M. elsdenii, P. ruminocola, S. ruminantium,* and *S. bovis*) and those in low numbers (1×10^7/mL ruminal fluid) with high activity (300 mmol $NH_3 \cdot min^{-1} \cdot mg^{-1}$ protein; *C. aminophilium, C. stricklandii,* and *P. anaerobius*). Ruminal protozoa contribute to the ammonia supply as well, directly by degrading ingested protein (Jouany, 1996) or perhaps indirectly by altering the bacterial profile that otherwise would exist in the absence of protozoa. Ruminal bacteria have a high affinity for ammonia and can survive on concentrations below 50 μ*M* (Schaefer et al., 1980), which means the fiber fermentation system is designed to survive episodes of low N supply yet function at ammonia concentrations ≥ 20 m*M*.

Rates and amounts of ammonia production reflect the solubility and fermentability of the dietary and endogenous sources of N. Sniffen et al. (1992) summarized data from a variety of feedstuffs. Forages, including corn silage, ranged from 7 to 28% of DM as CP. From 25 to 55% of CP was soluble in borate-phosphate buffer, and from 2 to 20% of CP was associated with ADF, and therefore considered to be unfermented. Nonprotein N comprised essentially all (most forages) or none (mature forages) of the soluble N. Grains and protein concentrates had a similarly wide range of N content and composition. Assuming 1) all of the soluble N and at least part of the insoluble N is fermentable and 2) a conservative estimate of endogenous N input (which also is fermentable) ranges from 12 to 41% of N intake (Table 1), one can conclude that at least half to essentially all of the N supply to the rumen enters the ammonia pool. Parker et al. (1995) used data from eight studies with sheep and calculated the linear relationship between N intake and irreversible loss of ruminal ammonia: Irreversible loss, g/d = 3.829 + .507(intake, g/d), $r^2 = .853$. The regression indicates that approximately 50% of N intake of the animals in these studies passed through the ammonia pool in the rumen and that the endogenous supply was 3.8 g/d. Therefore, it's not surprising that ruminal ammonia concentrations fluctuate by a factor of 5 to 8 (in a range from 3 to 45 m*M*) in animals consuming meals (Gustafsson and Palmquist, 1993; Robinson and McQueen, 1994; Horney et al., 1996; Rodriguez et al., 1997), with less (Shabi et al., 1998) to essentially no variation (Lana and Russell, 1997) as frequency of daily meals increases. Similarly, it is not surprising that ruminal ammonia concentration decreases in response to factors that diminish ammonia production, such as ionophores (Yang and Russell, 1993), or less-degradable N sources (Horney et al., 1996; Lana and Russell, 1997; Rodriguez et al., 1997), or factors that promote use of ammonia, such as synchronization of fermentable energy and N (Petit and Veira, 1994; Kolver et al., 1998; Olson et al., 1999).

Ammonia is absorbed, or diffuses, across all sections of the digestive tract of ruminants. Before diffusion across the gut epithelium the ammonium ion is converted to ammonia at the gut wall, diffuses into epithelial cells as ammonia, and then is protonated to re-form an ammonium ion. In addition to diffusion as ammonia, there are mechanisms that provide for transport of the ammonium ion, such as association with bicarbonate or VFA anions (Parker et al., 1995). The amount of N absorbed as ammonia is equivalent to 16 to 73% of N intake, which can be several times the amount of N absorbed as amino acids (Table 1). Absorption from the small intestine can account for 27 to 51% of net uptake of ammonia into the portal vein of cattle (Parker et al., 1995), which reflects both the potential for ammonia release in postruminal sections of the digestive tract and the efficient removal of ammonia from blood by the liver (Reynolds, 1992), thereby creating a concentration gradient that favors transfer from the lumen of the gut into blood. Ammonia is removed from blood by the liver. The transient, postprandial rise or chronic elevations in peripheral concentrations of ammonia may reflect splanchnic release of ammonia, or more likely reflects diffusion of ammonia directly from the digestive tract into peripheral blood vessels (Chalmers et al., 1971).

Basics of Urea Metabolism

Ruminants as well as other mammals synthesize urea, which helps prevent excess N from becoming toxic. Essentially all urea is produced in the liver, as discussed previously; however, other tissues have the enzyme activity required to urea production (Emmanuel, 1980). Once released into blood, urea is excreted in urine or reenters the digestive tract by diffusion into saliva or directly across the gut wall. The bulk of available data describing and quantifying sites and rates of urea metabolism in ruminants was available over 15 yr ago (see reviews of Kennedy and Milligan, 1980 and Huntington, 1986). The main point from that research is that urea production, excretion, and recycling to the gut are linked to diet composition, intake, and productive priorities of the animal. Depending on those factors, 19 to 96% of endogenous urea production may be recycled to the gut, 15 to 94% of the recycling may transfer in saliva, and 25 to 90% of urea degraded in the gut may be degraded in the postruminal digestive tract. Urea excreted in the urine represents from 25 to 60% of endogenous urea production in goats (Obara and Shimbayashi, 1980), sheep (Sarraseca et al., 1998), beef heifers (Bunting et al., 1989a), and beef steers (Huntington, 1989). Increased N intake increased the percentage of endogenous urea production that appeared in the urine of the goats and the beef steers but decreased that percentage in the urine of the beef heifers and sheep. In these studies, factors such as fermentability of dietary carbohydrate and N intake relative to nutrient

requirements affected the percentage of urea N excreted in urine.

Ureagenesis in the liver is closely linked to degradability of dietary N and subsequent absorption of ammonia (Tables 1 and 2). Ruminants, especially those consuming living or harvested legumes or immature grasses, depend on the liver to detoxify portal blood that contains ammonia absorbed from the gut. The capacity of the liver to perform that function is rarely exceeded, with instances usually associated with excess supply of dietary urea or similar rapidly degradable N source. The examples in Table 2 show a range from 45 to 386 g/d of urea N produced in the liver of cattle consuming from 0 to 480 g/d of dietary N. Table 2 also shows a high correlation between N intake (not including the feed-deprived steers) and liver production of urea N (r^2 = .96; liver production = .86 ± .07 times N intake) across a range of body weights and either high-forage or high-concentrate diets.

Urea is an important source of N entering the gut; that portion transferred directly across the gut wall is equivalent to 10 to 42% of N intake (Table 1, Huntington, 1986). Teleologically, N recycling provides a continuous source of ammonia to support microbial fermentation in the rumen as well as other regions of the digestive tract. Kennedy and Milligan (1980) listed ruminal ammonia concentration (negative relationship; ammonia decreases ruminal epithelium's permeability to urea), organic matter digestibility (positive relationship), and plasma concentration of urea (positive relationship) as the principal factors affecting rate of endogenous urea transfer from blood to the lumen of the gastrointestinal tract. Factors that affect capillary blood flow, such as CO_2 tension, may also affect urea transfer. Calculations of the percentage of recycled urea that was degraded in the bovine rumen range from 25 to 53%, with higher percentages in response to lower N intakes (Bunting et al., 1989a; Huntington, 1989) or higher intake of readily fermented carbohydrates (Huntington, 1989). In goats, increasing dietary N slightly increased the percentage degraded in the rumen from 43 to 46% (Obara and Shimbayashi, 1980). Plasma urea concentration and saliva production will affect how much urea enters the rumen with saliva; estimates of salivary contribution to endogenous ruminal urea transfer range from essentially all entering the rumen by transfer across the ruminal epithelium to approximately three times as much in saliva compared with direct transfer across the ruminal epithelium (Obara and Shimbayashi, 1980; Huntington, 1989; Huntington et al., 1996). Therefore, urea transfer can be altered by changes both in availability for transfer (supply, or "push") and in degradation of urea after transfer (use, or "pull"). Urea is rapidly hydrolyzed by bacteria adhering to ruminal epithelium or sloughed cells, and the resultant ammonia enters the ruminal ammonia pool (Bunting et al., 1989b). Amounts ranging from none to over 80% of ammonia from urea degradation are incorporated into bacterial N (Salter et al., 1979; Bunting et al., 1989a), and availability of energy is the major determinant of that percentage. In reality, the positive effects of organic matter digestibility and ruminal ammonia concentration on urea transfer are functions of the ruminal microbial capacity to assimilate products of fermentation.

Metabolism

The structure and function of the liver attests to the importance of removing potentially toxic ammonia from blood of ruminants as well as other mammals. The enzymes of the ornithine cycle and enzymes catalyzing transamination reactions are structurally oriented in mitochondria and cytosol of periportal and perivenous hepatic cells to form urea from ammonia absorbed from the gut and to use glutamine synthesis as another pathway to remove essentially all ammonia from hepatic portal blood (Meijer et al., 1990; Katz, 1992). Periportal cells remove ammonia from hepatic portal blood and use their enzymatic machinery to synthesize urea. The specialty of the perivenous cells is production of glutamine through glutamine synthetase, thereby providing another opportunity to remove ammonia from circulation before blood enters the hepatic veins and subsequently general circulation. This two-stage ammonia removal system integrates with other systems, including gluconeogenesis, regulation of acid-base balance, and interorgan N shuttles to derive the best metabolic control of substrate and product balances, nutrient supplies, and nutrient needs of the organism. Studies with multicatheterized sheep receiving an isotopically labeled infusion of ammonium chloride show that from 59 to 70% of urea N is derived from ammonia, with little effect of concentrate level in the diet (Lobley et al., 1995; 1996). Liver ureagenesis accounted for 13 to 19% of liver oxygen uptake.

As do other systems in the organism, N management systems respond to hormones and other metabolic regulators. Table 3 summarizes data from three studies with steers of similar body weight that provide examples of different scenarios and responses to changes in exogenous N supply. In all three studies, treatments included a control and abomasal infusion of casein to simulate increased postruminal supply of protein to the small intestine. The steers of Taniguchi et al. (1995; Table 3) received an additional 3.45 Mcal of DE in the form of acetate plus casein to augment the alfalfa hay in their control diet, whereas steers in the other two studies received only casein to supplement their high-grain diets. In all three studies, increased postruminal protein supply increased liver ureagenesis and recycling of urea into the gut. In two of the studies, the proportion of liver ureagenesis that was recycled through the portal-drained viscera increased, but in the study with dairy steers that proportion decreased, indicating increased priority of N salvage during the control period of that study. The study with dairy steers provides an example of the dramatic effect of exogenous somatotropin on reducing liver ureagenesis and increasing splanchnic release of amino acids. The effect of partitioning agents on liver N metabolism is also demonstrated in the data of Houseknecht et al. (1992) and Eisemann et al. (1993).

The magnitude of the response to casein infusion and the proportions recycled through portal-drained viscera vary in response to the amount of casein infused and amount of grain in the diet. In the study of Taniguchi et al. (1995; Table 3), 26 % of the increased N supply from casein could be accounted for by increased urea N released from splanchnic tissues, but in the other two studies that proportion exceeded 70%. However, examination of the main effect of site of casein infusion (ruminal or abomasal) balanced across additional energy from acetate shows that the site of casein infusion did not change the proportion of increased N supply accounted for by increased splanchnic release of urea N (Table 3), because the steers recycled additional liver ureagenesis that was the consequence of ruminal casein degradation.

The contribution of urea N to total urinary N in these examples (Table 3) provides some insight into the priority of excretion of excess N in relation to muscle mass, muscle growth, or perhaps acid-base regulation. The proportion of splanchnic to α-amino N present as glutamate provides some insight into potential changes in supply of essential amino acids in response to treatment, as well as priority of glutamate supply for tissue growth or interorgan N shuttles. However, one must remember that proportions respond to changes in the value of the numerator, the denominator, or both. When increased N supply was not accompanied by additional energy (acetate in the study of Taniguchi et al., 1995), the proportion of urea N in urine increased. The comparison of site of infusion of casein in steers fed alfalfa shows that the proportion of urine N present as urea increased slightly, even though the splanchnic supply of α-amino N almost doubled, the proportion of glutamate in splanchnic α-amino N was more than halved, total urinary excretion increased from 85 to 92 g/d, and N retention increased from 18 to 25 g/d. Combining exogenous somatotropin with increased postruminal supply of protein for the dairy steers (Bruckental et al., 1997) decreased the proportion of urine N present as urea N, increased the splanchnic supply of α-amino N, decreased the proportion of glutamate in splanchnic α-amino N, decreased urinary N from 51 to 40 g/d, and increased N retention from 46 to 58 g/d (Table 3). A converse pattern is seen in the third study; the data of Guerino et al. (1991) indicate that the steers converted the additional N to urea without major changes in splanchnic release of α-amino N or the proportion of that N as glutamate. In sum, these three examples show that both "push" and "pull" affect how the liver partitions N among urea, glutamate, and other amino acids.

Practical Applications

Three main areas of practical application of information relating to nonprotein N use by domesticated ruminants are 1) synchronization of carbohydrate and N fermentation in the rumen, 2) urea N as a convenient measure of protein status, and 3) N flow through ecological systems. Synchronization of ruminal fermentation has the attractive outcomes of improved fiber digestion, increased output of microbial biomass (with emphasis on microbial protein), and decreased absorption of ammonia. Elizalde et al. (1999) added fermentable carbohydrate to a diet of fresh alfalfa that contained large amounts of rapidly available N; this decreased ruminal ammonia levels and increased duodenal supply of amino acids in steers. Similarly, adding starch to a low-quality hay supplemented with degradable protein (casein) decreased ruminal ammonia levels but it also reduced fiber digestion and voluntary forage intake of steers (Olson et al., 1999). Recent efforts to improve productive performance of dairy cows through improved synchronization of carbohydrate and N fermentation in the rumen successfully altered ruminal ammonia levels and capture of N as microbial protein but did not result in detectable improvement in performance (Robinson and McQueen, 1994; Kolver et al., 1998; Shabi et al., 1998). We calculate from data of Lobley et al. (1996) that from 2.5 to 5% of whole-body oxygen consumption (energy lost as heat) can be attributed to ureagenesis in the liver, based on the "high" estimate of 4 mol of ATP used for every mole of urea produced (that may be as low as 1 mol of ATP per mole of urea produced). Therefore, changes in ureagenesis that come from changes in ammonia absorption from rumen fermentation will cause changes in ME use that are difficult to detect.

Dijsktra et al. (1998) reviewed concepts and mathematical models that evaluate and predict ruminal energy and N fermentation kinetics. The relatively simple concept of synchronizing fermentation of N and carbohydrate sources in the rumen is complicated by several associated factors, including rates of voluntary intake, rates of passage of liquid and dry matter from the rumen, transfer of metabolites and fermentation products across the rumen wall, adaptation of rumen microbiota to the current conditions, and interactions that affect efficiency of microbial growth. Their discussion of recycling of urea into the rumen points out that few, if any, models take a mechanistic approach to this recycling and attribute importance to recycling only if ruminal degradable protein intake is low. For example, the CNCPS model (Russell et al., 1992) uses an equation from NRC (1985) to predict urea recycling as a negative linear and positive quadratic function of CP intake. Dijkstra et al. (1998) concluded a discussion of urea recycling by stating, "A holistic approach, providing for relevant interactions between microbial metabolism in the rumen and animal metabolism, would be more appropriate."

Concentrations of urea N in blood, plasma, serum, urine, or milk have been recognized for several years as useful measures of (changes in) protein status or protein nutrition of ruminants (Hammond, 1983; Hof et al., 1997). Jonker et al (1999) developed a model to predict urinary N excretion as the first step in predicting milk urea N concentrations. These predictions allow dairy producers to evaluate adequacy of their cows' nutrition program. Although the model is not as sensitive as desired to factors such as body weight, parity, or grouping strategy, it represents a mecha-

nistic approach to the use of milk urea N concentrations as a management tool.

Until the last half of the 20th century, production efficiency was the major, if not the only, decision criterion for nutrition of domesticated ruminants. Increased urbanization and enhanced intensive technologies have added environmental or ecological concerns as additional, and perhaps major, decision criteria. Capture of N as product (milk, wool, hair, meat, and offspring) is less than 30% of total N input, with higher percentages associated with lactation and percentages of 5% or less associated with grazing ruminants (Van Horn et al., 1996). Urine and fecal excretion account for almost all remaining N input (Tamminga, 1992), and although some of that excretion may be used by plants to produce biomass that is removed as crop or recycled to ruminants and other herbivores, much of excreted N enters the atmosphere and ground water, thus becoming a potential pollutant. Therefore, past latitude in excess nutrient supply will give way to closer calculation of diet composition and intake, perhaps with a cost associated with nutrient excretion factored into the formulation (Tamminga, 1996; Van Horn et al., 1996).

Implications

Ruminant digestive and metabolic systems accommodate a wide range of feedstuffs. A major component of that accommodation is integrated metabolic management of nonprotein nitrogen. Ruminants can survive on low-protein feedstuffs by recycling urea nitrogen to support rumen function yet can tolerate absorption of large amounts of ammonia when their diets contain lush, immature forages. Practical feeding systems of the future will use ruminants' versatility to apply the best combination of priorities from profitable animal production, optimal use of available feed resources, and sustenance of desired ecological systems.

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Notes

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Table 1. Nitrogen intake, digestibility, retention and net flux across portal-drained viscera of cattle (from Huntington, 1999)

		Percentage of N intake				Net flux across portal-drained viscera, % intake		
Animal	Diet	N intake, g/d	Digested	Excreted in urine	Retained	Ammonia N	Urea N	α-Amino N
Lactating cow[a]	Corn silage:supplement 60:40	388	69	45	–13.7	47	37	36
Dairy steer[b]	Alfalfa silage	193	72	58	14	73	15	18
	Alfalfa silage	273	71	57	14	57	15	19
	Orchardgrass silage	157	60	53	7	57	20	24
	Orchardgrass silage	198	61	46	15	24	19	26
Beef steer[c]	Alfalfa hay	119	74	60	13	56	12	21
	Alfalfa hay, ruminal casein	150	77	61	15	62	20	14
	Alfalfa hay, abomasal casein	150	74	57	17	46	15	24
Beef steer[d]	Hay:concentrate 75:25	86	75	56	19	54	34	20
	Hay:concentrate 75:25	159	72	56	17	44	30	28
Beef heifer[e]	Hay:concentrate 75:25	133	70	64	6	47	28	24
	Hay:concentrate 75:25	209	67	58	9	69	34	36
	Hay:concentrate 25:75	98	74	60	15	48	39	36
	Hay:concentrate 25:75	174	70	56	14	54	34	20
Dairy steer[f]	72 % Concentrate	123	58	31	27	31	36	34
	72 % Concentrate, abomasal casein	144	68	36	32	36	41	53
Beef heifer[g]	10:90 Hay:concentrate	34	70	33	37	35	20	28
	10:90 Hay:concentrate	64	67	33	33	21	23	30
	10:90 Hay:concentrate	93	63	33	30	16	19	33

[a]Reynolds et al., 1988 ; [b]Huntington et al., 1988 ; [c]Taniguchi et al., 1995; [d]Reynolds et al., 1992 ; [e]Reynolds et al., 1991a,b ; [f]Bruckental et al., 1997 ; [g]Huntington and Prior, 1983,1985.

Table 2. Net flux[a] of ammonia N (NH_3), urea N, and α-amino N (AAN) in cattle

Animal	n	BW, kg	Diet	N intake, g	Net PDV flux, g/d			Net liver flux, g/d			Reference
					NH_3	Urea N	AAN	NH_3	Urea N	AAN	
Beef steer	6	416	3-d Feed deprivation	0	26	–13	1	–34	61	–29	Eisemann and Nienaber, 1990
Beef steer	4	253	Alfalfa hay	119	67	–14	25	–68	89	–18	Taniguchi et al., 1995
Beef steer	6	421	73% Switchgrass	100	41	–35	32	–42	45	–16	Huntington et al., 1996
Dairy cow	4	NA[b]	Ryegrass	380	76	–102	NA[b]	–119	259	NA	de Visser et al., 1997
			Ryegrass	480	241	–127	NA	–234	386	NA	
Dairy steer	6	289	72% Corn	123	38	–44	42	–51	59	–29	Bruckental et al., 1997
Beef steer	6	445	70% Corn	210	68	–31	47	–73	95	–39	Eisemann et al., 1996
		319	70% Corn	162	71	–22	40	–74	80	–23	
		236	70% Corn	128	48	–17	31	–51	50	–11	

[a]Positive flux is absorption or release; negative flux is transfer or uptake.
[b]NA = not available.

Table 3. Nitrogen intake (NI), urinary excretion, and net flux[a] of urea N and α-amino N (AAN) across portal-drained viscera (PDV), liver, and total splanchnic (TSP) tissues of steers in response to increased duodenal protein supply or exogenous bovine somatotropin (BST)

			Urea N, g/d						
Animal	Treatment	NI, g/d	PDV[d]	Liver	TSP	Urine	Urine urea N/urine total N	TSP AAN, g/d	TSP glutamate/TSP AAN
Beef steer [b]	Control	96	–16(.28)[e]	58	42	14.5	.64	21.8	.37
	Abomasal casein	135	–32(.35)	92	60	16.7	.77	20.2	.42
Beef steer [c]	Control	119	–14(.16)	89	76	57	.79	6.7	.36
	Abomasal casein	150	–22(.21)	105	83	65	.77	14.1	.38
	Ruminal casein	150	–30(.27)	112	82	69	.75	7.7	.83
Dairy steer [d]	Control	123	–44(.75)	59	15	38	.57	13.1	.80
	Abomasal casein	144	–59(.58)	102	43	51	.69	18.3	.58
	Abomasal casein and BST	144	–40(.59)	68	28	40	.66	43.6	.36

[a]Positive flux is absorption or release; negative flux is transfer or uptake.
[b]Guerino et al. (1991). Seven steers (BW = 290 kg) fed a high-grain diet and infused abomasally with water (Control) or 300 g of casein daily.
[c] Taniguchi et al. (1995). Four steers (BW = 253 kg) fed alfalfa hay (Control) or infused with 800 g/d of cornstarch and 200 g/d of casein into the rumen or into the abomasum.
[d]Bruckental et al. (1997). Six Holstein steers (BW = 254 kg) fed a high-grain diet (Control), infused abomasally with 300 g/d of casein or infused with casein and 20 mg/d of recombinant BST.
[e]The number in parentheses is the proportion of liver ureogenesis that was recycled through the PDV.