REVIEW: Urea Feeding to Dairy Cattle: A Historical Perspective and Review

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ABSTRACT

Urea has been fed in ruminant rations for more than 100 yr. Its use in dairy cattle rations has fluctuated with protein and urea prices, with various values used in different formulation systems, and with mixed to negative experiences in experiments and field situations. In many studies, rations were not isocaloric when urea was added, and intake reduction occurred because of high dietary levels of urea. Some studies concluded that cows disliked the flavor or odor of urea, or that there was some metabolic component. A series of studies revealed that cows did not dislike the flavor or odor of urea per se, that they could identify different levels of urea in rations, that they developed a conditioned negative aversion to urea when urea was fed at higher levels and for several exposures, and that 135 g/cow daily did not reduce DMI. In older studies, and in even more recent ones, this level of urea intake has been exceeded by 50 and up to 300% with a corresponding DMI decrease, even when fed in TMR. Urea use has also been limited because of in vitro studies showing no benefit to adding urea if ammonia levels are at 5 mg/mL or dietary CP is at 13%. However, several in vivo and in situ studies have shown the optimal rumen ammonia level to be between 17 and 25 mg/mL when DM disappearance and nonammonia-N flow are the determining measurements. Several studies have shown that oils, especially more unsaturated oils, defaunate or reduce protozoa, which can increase microbial protein synthesis efficiency but reduce DMI. In one study, the authors speculated that addition of urea could be beneficial to counteract reduced rumen ammonia and pH. Although there is some belief that addition of urea to higher nitrate-containing diets exacerbates the situation, studies do not support this contention. A large-scale field study and a long-term feeding study did not show any meaningful negative effects on reproduction when feeding urea. Synchronizing rumen N available with carbohydrate fermentation has a theoretical benefit, but a recent review found this did not occur, most likely because of N recycling and because of the adaptability of rumen microorganisms to asynchronous N and energy supply. Various commercially processed urea products have been developed, but few studies have been published showing that the processing and feeding objectives were achieved. Adding urea sources to ensiled forages has increased final N content and reduced protein degradation of the silage. When urea was also added in the concentrate, no negative effects were seen if total supplemental NPN was less than 20% of total dietary CP. Classic ammonia toxicity from too much dietary urea being provided in a short period is most closely related to rumen pH because urea hydrolysis elevates rumen pH, which then allows more rapid absorption of the now greater levels of rumen ammonia into the blood. Traditional recommendations for feeding urea to dairy cows have been excessive. More reasonable recommendations would be for not more than 1% in the concentrate, approximately 135 g/cow daily, and not more than 20% of total dietary CP coming from added urea-NPN sources.

Key words: dairy cattle, feeding, intake, level, urea

INTRODUCTION

German workers (Ehrenberg et al., 1891; Zuntz, 1891) determined that urea could be used to replace a portion of protein in ruminant rations. As recently as 1937, it was not widely recognized that urea is converted to proteins in amounts of any great significance to ruminants (Reid, 1953). Reid (1953) concluded from his extensive literature review that 1) conversion of urea to protein is mediated by the microorganisms of the rumen and reticulum, which subsequently avail the host animal of their protein content; 2) a low level of protein and high level of starch in the ration favor urea utilization; 3) bacteria may prefer highly soluble and readily hydrolyzable protein rather than urea in the ration; 4) sugars and cellulose are
inferior to starch as sources of energy for ruminal microorganisms; 5) application of in vitro to in vivo experiments may be misleading because the characteristics and kinds of microorganisms may be different in vitro after even relatively short periods; 6) older calves have faster growth rates with urea, whereas calves as young as 2 mo of age have been shown to use some urea-N; 7) addition of methionine or S has improved the retention of N by lambs fed urea-containing rations; 8) rendering urea hydrolysis more slowly to minimize ammonia wastage may be a fruitful approach; 8) urea is somewhat inferior for dairy and beef calves fed rations containing 12% or more of protein equivalent, of which three-fourths of the N is supplied by conventional protein sources; 9) a level of 1% urea in the concentrate ration of fattening calves may be unpalatable and may reduce feed intake; 10) urea may provide up to 25% of the N in rations containing 12% protein equivalent for fattening lambs and for pregnant or lactating ewes; 11) urea N may provide up to 27% of required N from the standpoint of milk yield or reproductive behavior or general health; 12) urea may provide up to 3% of the concentrate ration or up to 1% of the total ration for milking cows from a practical standpoint; 13) small quantities of urea undiluted by feed (116 g in cattle and 10 g in sheep) and introduced suddenly into the rumen resulted in rapid onset of toxicosis, whereas 180 to 272 g urea was consumed daily by beef calves or cows without toxicosis when fed along with hay or corn silage; 14) cattle refuse to consume enough feed to be harmed because of the unpalatability of urea; 15) when urea is fed at satisfactory levels affecting protein replacement, palatability does not appear to be noticeably reduced; 16) molasses may improve palatability of urea-containing rations; and 17) because urea has no energy value for animals, feeds containing urea must be fed at a slightly higher rate to provide both N and digestible nutrients equivalent to those provided by conventional feed. This review addresses studies conducted since the 1953 review by Reid, and is directed toward urea use in dairy cattle diets.

Feed Use

Estimated amounts of annual US feed-grade urea use [Allen and Devers (1975), and industry estimates] provide insights into factors influencing urea use for ruminants (Figure 1).

Urea Use Formulation Systems

In 1973, another significant event occurred with the publication in a popular dairy magazine of an article stating that cows producing more than 22.7 kg milk/cow daily could not utilize urea when the ration already contained 12% protein (Roffler and Satter, 1973). During that same period, it is likely that there were numerous negative experiences by many dairies that had felt forced to use urea because of the very high protein costs. When these dairy managers also read that their good cows could not use urea, and when this idea was reinforced by many others who had adopted that recommendation, the negative image of urea was further reinforced. This was countered by some researchers (Bartley, 1976; Huber, 1976a,b; Conrad, 1977) not in concert with the recommendations of Roffler and Satter (1973), but their work was viewed with some suspicion because they were involved with the following processed urea-containing products: Starea, Dehy-100, and ProSil, respectively.

In the early 1970s, several other systematic approaches to formulation emerged, which further restricted or eliminated the use of urea. In the soluble protein system (Sniffen, 1974), urea was considered to be a 100% soluble N source, whereas in the urea
fermentation potential system (Burroughs et al., 1975), urea use was tied to the energy fermentation potential of the ration. The latter reference was part of a 1974 American Society of Animal Science symposium on Protein Physiology and Its Applications in the Lactating Cow. Other presentations published from that symposium were those by Huber (1975) and Kertz and Everett (1975). Although the presentation at that symposium by Roffler and Satter (1974) was not published, much of that presentation appeared in another publication (Satter and Roffler, 1975).

**Dairy Production Trials—Reduced DMI with Higher Urea Level and Lower Energy Level**

The admonition by Reid (1953) to have equivalent N and digestible nutrient intakes when using urea in rations was not always accomplished. This was due to dairy cows often reducing their intake with urea-containing rations and because the energy density of rations was not always being adjusted to compensate for the nonenergy contribution by urea. This was particularly problematic before the 1970s, when most dairy cows were fed the concentrate primarily in milking parlors, where they were milked twice daily. This feeding practice limited the time cows had available to eat, and did not have the advantage of further diluting urea concentration with forage, which accompanied the later adaptation of TMR. The problem of limited eating time was minimized by Plummer et al. (1971) by feeding cows their concentrate only in a stanchion barn and by gradually switching cows from nonurea to urea-containing concentrates. While adding urea at 2 and 3% of concentrate and keeping the N content equivalent, TDN was decreased from 75 to 73.5 to 72.8% for the no-urea control, 2% urea-containing concentrate, and 3% urea-containing concentrate, respectively. Intake and milk production did not differ among rations, but milk fat percentage and the ratio of acetic to propionic ruminal VFA increased with the use of 2 or 3% urea. Plummer et al. (1971) contrasted these results with a trial by Van Horn et al. (1967) in which concentrates contained no urea, 2.2% urea, or 2.7% urea, with corresponding calculated TDN of 77.9, 76.5, and 69.4%, respectively. In that trial, each cow was offered 4.6 kg hay, 18.2 kg corn silage, and 18.2 kg concentrate daily. Cows were overfed on concentrate to assess the relative acceptability of the respective mixtures, but daily allowances were divided into 3 portions of 6.8, 4.6, and 6.8 kg, distributed during the day, with hay and silage fed twice daily. Despite this feeding regimen, intake of urea-containing concentrates was decreased ($P < 0.01$) along with a corresponding decrease ($P < 0.01$) in milk production. Daily urea intake was 235 g for 2.2% urea-containing concentrate and 211 g for 2.7% urea-containing concentrate. These urea intakes contrasted with those found in a summary by Van Horn et al. (1967) of 12 experiments reporting nonsignificant differences in milk production, which also involved comparisons of 22 urea-containing rations with isonitrogenous control rations. Of the 22 comparisons, cows on urea produced less than the control cows 15 times, the same as control cows 3 times, and more than the control cows 4 times. The average depression in milk production was 1.7%, with an average urea intake of approximately 136 g daily. Most of the cows were Holsteins.

Polan et al. (1976) used a multifactorial approach in which 9 total rations contained either 9.4, 11.1, 12.8, 14.5, or 16.2% CP, with urea supplying 0, 10, 20, 30, or 40% of CP. Concentrations of urea were 0, 1.0, 1.3, 1.4, 2.4, 3.1, 4.3, and 5.1% in concentrates, which constituted 38% of total DMI, with the balance coming from corn silage. Although these rations were described as isocaloric, they ranged in calculated TDN from 73.9 to 69.4%, which was inversely related ($r = -0.96$) to urea level. Although Figure 2 was cited as supporting the concept of Roffler and Satter (1975a,b), DMI was the major factor (Figure 3), as influenced by urea and CP levels. Although Figure 2 was cited as supporting the concept of Roffler and Satter (1975a,b), DMI was the major factor (Figure 3), as influenced by urea and CP levels. This is best illustrated by Figure 4, in which within CP level (especially at greater CP levels, which also had greater urea levels), greater levels of urea depressed both DMI and milk production.

![Figure 2. Milk production response to dietary protein and urea (Polan et al., 1976; used by permission).](image-url)
Urea Intake Depression Mechanism

In the early to mid-1970s, several studies were done to decipher whether the reduced intake with dietary urea was due to taste, odor, or metabolism. Although at that time it was generally assumed that cows did not like the taste of urea, this did not explain how cows initially exposed to higher levels of urea in a ration consumed enough to cause death (Anonymous, 1974).

Huber and Cook (1972) fed 4 ruminally fistulated cows 5.4 kg of alfalfa hay once daily and concentrate ad libitum twice daily. Treatments were concentrate containing 1% urea (control), 3% urea, 1% urea + 2% urea equivalent (ruminally dosed), and 1% urea + 2% urea equivalent (abomasally dosed). Because only 3% urea decreased concentrate intake, the data were interpreted to mean that cows objected to the taste of urea, and there were no ruminal or postruminal effects. However, eating time was not restricted, nor was eating rate observed.

In another trial (Huber and Cook, 1972), 3.5% urea was used in concentrates containing 7.5% molasses and 7.5% beet pulp mixed by 2 different methods. Considerable variation resulted, with intake being nominally lowest for the concentrate without beet pulp, but the authors acknowledged that this method was not satisfactory for rapidly identifying unpalatable concentrates. Van Horn et al. (1967) also found that adding 4.7% molasses to a concentrate containing 1.9% urea did not prevent intake depression compared with a nonurea concentrate.

Wilson et al. (1975) went farther in defining some of the parameters leading to intake depression with urea. Four ruminally fistulated Holstein cows were fed rations containing 33 to 35% cottonseed hulls as the roughage source and 1.0, 1.65, 2.3, or 3.0% urea in a 4 × 4 extra period Latin square design. They were fed ad libitum for the first week, after they had been adapted to the urea-containing rations the previous 2 wk. During the second week, cows were fed only the 1% urea-containing ration, with the balance of urea greater than 1% added directly into the rumen 3 times daily in a 20% aqueous solution. Intake decreased with increasing level of urea in rations for both oral and rumen treatments. Daily urea intakes were considerably beyond the 136 g daily average established by Van Horn et al. (1967) from their analysis of 22 comparisons.

A second trial was also a 4 × 4 Latin square with weigh-backs recorded daily at 15 min before feeding and then at 30-min intervals for 1.5 h after feeding. At each feeding, a 20% solution of urea was administered directly into the rumen. The urea administered daily amounted to 2% of the ration consumed the previous day. Urea-containing rations fed orally had greater intakes than when urea was infused or poured into the rumen. Most of this difference occurred during the first 30 min after feeding. Infusing urea continuously over 2.5 h decreased intake less than when urea was poured into the rumen twice daily. Wilson et al. (1975) concluded that 1) the taste of urea was not the sole cause of intake depression, 2) greater
than 1% urea depressed intake, and 3) metabolic intermediates of urea catabolism may have accounted for part of the intake depression when more than 1% urea was fed.

Because it was then commonly assumed (and often still is) that the ammonia odor from urea caused the initial rejection of urea-containing rations, ammonia concentrations of 40, 181, and 462 mg/kg were created with feed boxes immediately before cows were allowed to consume the nonurea ration with or without ammonia in a 2-choice test for 30 min twice daily (Kertz et al., 1977). Although cows exhibited increasingly reactive nasal symptoms on initial exposure to greater ammonia concentrations, this did not alter their subsequent consumption of the nonurea rations either with or without the presence of the ammonia odor. Consequently, the ammonia odor per se did not appear to be causing the initial rejection of urea-containing rations.

A series of palatability studies were then designed to include urea in various parts of the pellet to determine whether cows disliked the taste or odor of urea (Kertz et al., 1982). All urea-containing rations had 2.5% urea, regardless of how it was packaged. Whereas cows regularly chose to consume more of the nonurea rations versus the 2.5% urea-containing rations, they also clearly chose to eat more of the urea-containing rations in which urea was most exposed to taste or odor, when given a choice between 2 urea-containing rations. Thus, cows did not dislike the taste or odor of urea per se. But how, why, and when did cows discriminate against urea-containing rations?

It was known previously (Kertz and Everett, 1975) that cows did not discriminate against rations containing 1% urea. Four ruminally fistulated cows were monitored for intake every 5 min for 30-min feeding periods twice daily (Kertz et al., 1982). Cows detected and reduced their intake within the first 5 min for the 2.5% urea-containing ration compared with intakes of both a 1% urea-containing ration and a nonurea ration, which were similar. This was most pronounced for the morning feeding because it had been 18 h since the previous afternoon feeding. The short 6-h period between the morning and afternoon feedings reduced afternoon intakes and differences among rations.

At the same time, the rumens of cows in the eating rate study were sampled for pH and ammonia analyses (Table 1) before and immediately after each morning feeding on eating rate measurement days. Intake was decreased with the 2.5% urea-containing ration. Rumen pH did not decrease after feeding the 2.5% urea-containing ration, decreased slightly for the nonurea ration, and decreased immediately for the 1% urea-containing ration. Both urea-containing rations resulted in increased rumen ammonia concentrations after feeding. However, if subclinical ammonia toxicity elicited a decrease in intake of a urea-containing ration, then 2 events had to occur: subclinical ammonia toxicity and a mechanism by which the cow could identify that event with the feed, resulting in intake reduction.

The pKa of ammonia is 9.02 (Visek, 1968). Ammonia must be in the ionic form of ammonium (+NH₄⁺) to be absorbed across the rumen wall into the blood. In the acidic environment of the rumen, ammonia is essentially completely ionized. As the pH increases into the upper 6.0 range, ammonia absorption increases (Visek, 1968). Data from studies by Bartley et al. (1976), Visek (1968), and Smith (1975) indicated that rumen pH at or greater than 7.0 greatly facilitated the absorption of ammonia. The classical toxicity studies of Bartley et al. (1976) demonstrated that rumen pH had a higher correlation (r = 0.317) with toxicity than rumen ammonia-N (r = 0.039). That is because rumen pH determines how rapidly and how much ammonia is absorbed into the blood. Consequently, blood ammonia-N had the highest correlation (r = 0.707) with toxicity.

Although both urea treatments in Table 1 had high and similar postfeeding ammonia levels, rumen pH was not as high (P < 0.05) with the 1% urea-containing ration as with the 2.5% urea-containing ration. As urea is hydrolyzed to ammonia, it has 2 effects. Rumen ammonia is increased, but as ammonia is ionized to ammonium with the addition of 1 H ion per molecule, rumen pH is elevated or not depressed as greatly, depending on the amount of urea hydrolyzed, other dietary factors, and rumen microbial activity. Consequently, it appeared that 1% urea did not create subclinical ammonia toxicity, whereas 2.5% urea did, as supported by differences in feed intake (Table 1). This led to a study (Kertz et al., 1983) in which ammonium chloride was dosed into the rumen of cows at urea-equivalent levels. The amount dosed was equivalent to the ammonium that would have been derived from the urea had it been included in the nonurea rations formulated without 1 or 2%

Table 1. Rumen parameters relative to the morning feeding

<table>
<thead>
<tr>
<th>Ration</th>
<th>Daily intake, kg/cow</th>
<th>pH Before</th>
<th>pH After</th>
<th>Ammonia, mg/100 mL Before</th>
<th>Ammonia, mg/100 mL After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonurea</td>
<td>12.6</td>
<td>6.90a</td>
<td>6.27b</td>
<td>23.7a</td>
<td>37.5b</td>
</tr>
<tr>
<td>2.5% urea</td>
<td>9.2</td>
<td>6.82</td>
<td>6.82a</td>
<td>26.0a</td>
<td>107.5b</td>
</tr>
<tr>
<td>1.0% urea</td>
<td>13.6</td>
<td>6.78a</td>
<td>6.44b</td>
<td>26.9a</td>
<td>106.7b</td>
</tr>
</tbody>
</table>

a,bMeans with different lowercase superscripts within a row within pH or within ammonia differ (P < 0.05).

a,bMeans with different uppercase superscripts within a column differ (P < 0.05).

1From Kertz et al. (1982).
urea. Because ammonium chloride simply dissociates in the rumen, it would not have the rumen pH elevating effect of urea hydrolysis. Although ammonium chloride considerably increased rumen ammonia (Table 2), because rumen pH was not elevated, as occurs with urea hydrolysis, the rumen ammonia was essentially trapped, resulting in no significant increases in blood urea N and ammonia and no decreases in feed intake.

**Conditioned Negative Aversion**

The missing link was how cows learned to reduce their intake of high urea-containing rations. Seven first-calf heifers without any previous exposure to urea-containing rations were purchased from a local dairy (Kertz et al., 1982). After these cows had passed peak lactation while being fed nonurea rations, they were assigned a sequence of urea-containing rations fed during the week, with a nonurea ration fed on 2-d weekends (Table 3).

Intake variation on weekends was very low (7.2% CV), so there were differences ($P < 0.05$) among weekend intakes. The general trend was for lower intakes toward the end of the study, except for d 22 to 23. Intake was lower over the last several weekends because of the hot and humid August weather. Although initial exposure to 2.5% urea did reduce intake by 13%, that was only approximately one-half to one-third of the reduction seen in most of the previous studies (Kertz et al., 1982). The 1 and 1.5% urea-containing rations did not reduce intake. In addition, greater ambient temperatures affected intake over the last 3 wk. Although 2% urea reduced intake relative to nonurea for d 3 to 7, it was similar to the nonurea intake for d 45 to 49. The 2.5% urea-containing ration reduced intake from d 38 to 42, but not to the extent previously seen in other studies (Kertz et al., 1982).

At the end of the trial (Table 3), three of these cows were used to evaluate a load cell recorder for several days (Kertz et al., 1982). Combined morning and afternoon intakes of the 2.5% urea-containing ration (7 kg) were 26% less than the weekend nonurea ration consumption (9.7 kg) when the entire ration fed was consumed. In the afternoon feeding, after cows stopped consuming the 2.5% urea-containing ration, they were offered the nonurea ration for the remainder of the 30-min feeding period. Their intake of the nonurea ration (3.23 kg) nearly equaled the consumption of the 2.5% urea-containing ration (3.57 kg), indicating that cows readily identified the ration as not containing urea and rapidly resumed consumption. Thus, it appeared that one or more exposures to high dietary levels of urea are required to produce a subclinical ammonia toxicity, which the cows then learn to associate with the urea-containing ration, reducing their intake to prevent the reoccurrence of subclinical ammonia toxicity. This is a classical case of conditioned negative aversion. This was independently confirmed in a study with Holstein heifers (Chalupa et al., 1979). In all these studies, a daily urea intake of 135 g/cow never resulted in

| Table 2. Mean rumen and blood parameters at 0, 0.5, 1.5, 3.0, and 6.0 h after feeding
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Treatment</td>
<td>Ration intake, kg/cow per day</td>
<td>Rumen</td>
<td>Blood</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pH</td>
<td>Ammonia, mg/100 mL</td>
</tr>
<tr>
<td>No ammonium chloride</td>
<td>8.8</td>
<td>5.63</td>
<td>17.3&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ammonium chloride equal to 1% urea</td>
<td>10.1</td>
<td>5.67</td>
<td>101.7&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Ammonium chloride equal to 2% urea</td>
<td>9.0</td>
<td>5.71</td>
<td>154.9&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
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<td>&lt;sup&gt;a,b&lt;/sup&gt;Means within a column with different superscripts differ ($P &lt; 0.05$).</td>
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<td>1From Kertz et al. (1983).</td>
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<p>| Table 3. Ration consumption by weekends and during a 5-d wk |</p>
<table>
<thead>
<tr>
<th>Weekend day</th>
<th>Nonurea ration intake, kg/cow per day</th>
<th>Day</th>
<th>Ration</th>
<th>Ration intake, kg/cow per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 2</td>
<td>12.9&lt;sup&gt;abc&lt;/sup&gt;</td>
<td>Weekends</td>
<td>Nonurea</td>
<td>12.3&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>8 to 9</td>
<td>12.6&lt;sup&gt;abc&lt;/sup&gt;</td>
<td>3 to 7</td>
<td>Nonurea</td>
<td>12.9&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>15 to 16</td>
<td>13.0&lt;sup&gt;b&lt;/sup&gt;</td>
<td>10 to 14</td>
<td>2.5% urea</td>
<td>11.2&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>22 to 23</td>
<td>11.9&lt;sup&gt;c&lt;/sup&gt;</td>
<td>17 to 21</td>
<td>1.0% urea</td>
<td>12.9&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>29 to 30</td>
<td>13.3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>25 to 28</td>
<td>1.5% urea</td>
<td>13.2&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>36 to 37</td>
<td>12.0&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>31 to 35</td>
<td>2.0% urea</td>
<td>11.1&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>43 to 44</td>
<td>12.1&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>38 to 42</td>
<td>2.5% urea</td>
<td>9.4&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>50 to 51</td>
<td>10.8&lt;sup&gt;c&lt;/sup&gt;</td>
<td>45 to 49</td>
<td>Nonurea</td>
<td>11.0&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>&lt;sup&gt;a,b&lt;/sup&gt;Means with different superscripts within a column differ ($P &lt; 0.05$).</td>
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<tr>
<td>1From Kertz et al. (1982).</td>
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Dairy Production and Urea Utilization Trials

Earlier production trials using urea were often confounded by DMI decreases and lower ration energy levels, as noted before. A study by Clark et al. (1973) used 0, 1.6, and 2.4% urea in concentrates in the first trial, 2.42 and 2.83% in the second trial during the first 100 d in lactation, and 1.8 and 2.1% in the second trial later in lactation. Daily DMI was low, at 12 to 14.4 kg; daily 4% FCM was low, at 13.7 to 18.1 kg; and urea intake levels were high, at 146 to 204 g in trial 1 in early lactation and at 167 to 178 g in trial 2 in early lactation. Another experiment fed TMR (Kwan et al., 1977) with 1) a negative control of 11.7% CP, 2) 13.9% CP with 1% urea, 3) 16.6% CP with 1% urea, or 4) a 16.6% CP positive control with no urea for 12 wk, beginning 5 wk postpartum. Dry matter intake was the lowest (P < 0.05) for the nonurea negative control and was highest for the high-protein diet with 1% urea. The former would be expected because of the effect of lower CP concentration limiting DMI. The latter was mainly a function of body size because more large cows happened to be randomly assigned to the 16.6% CP with 1% urea diet. Daily urea intake averaged 172 to 203 g/cow. Milk production was greater (P < 0.05) for all higher CP diets, and tended to be the greatest for the 16.6% CP with 1% urea diet. Cows on the 16.6% CP with no urea diet were younger by 4 to 10 mo. Milk fat percentage was similar among treatments. Rumen ammonia was lowest, even below the 5 mg/100 mL recommendation of Satter and Roffler (1975), for the lowest CP nonurea negative control diet. These samples were taken 2 h postfeeding from each of 3 ruminally fistulated cows per treatment. Dry matter digestibility was higher (P < 0.05) for all treatments compared with the negative control. Nitrogen balance also tended to be higher for these diets. The authors concluded that milk production responses, N balance data, rumen ammonia, and plasma N metabolism data all supported the view that these lactating cows effectively used added NPN (urea) in diets of 13.9% and probably above this amount.

Broderick et al. (1993) fed alfalfa silage containing 60% DM and 25.5% of the diet along with 30.1% corn silage for cows in early lactation in a 4 × 4 Latin square for trial 1. Diets were isonitrogenous at 16.3% CP and differed by 1.33% urea in one diet, or by various combinations of nonurea ingredients (soybean meal, meat and bone meal, or soybean meal + meat and bone meal) as protein supplements in the other diets. Net energy for lactation estimates were 2.4% lower for the urea-containing diet. Cows averaged 33 kg/d of milk and 25.1 kg/d of DMI, which did not differ among treatments. Yields of milk components and concentrations of free amino acids in plasma were similar. Average daily urea intake was 338 g, but TMR diets were used, which diluted the urea concentration throughout the entire diet when fed. In trial 2, cows were fed alfalfa silage at low (39%) or high (59%) DM at approximately 27% of dietary DM along with a similar amount of corn silage. Diets were isonitrogenous at 16.3% CP, but NE estimates were approximately 2.7% lower with 1.63% urea as compared with diets supplemented with soybean meal + meat and bone meal. Dry matter intake was reduced (P < 0.05) for the urea-containing diet with low-DM alfalfa silage, but not for the diet with high-DM alfalfa silage. Daily urea intakes were, respectively, 397 and 419 g, 20% more than in trial 1. Cows averaged 37 kg/d of milk and 25.6 kg/d of DMI. Compared with urea, soybean meal + meat and bone meal with low-DM alfalfa silage increased (P < 0.05) the yield of milk and milk components. Lower (P < 0.05) concentrations in trial 2 of ruminal ammonia and urea in milk and blood, accompanied by higher (P < 0.05) rumen pH, indicated lower degradability of the true protein meals and an excessive level of urea feeding under these trial conditions.

Brito and Broderick (2007) followed up with a study in which alfalfa silage provided 20.7% of DM, corn silage provided 35% of DM, and high-moisture shelled corn provided from 40.7 down to 26.5% of DM for TMR diets in which the CP treatments were 1.9% urea, 12.1% soybean meal, 14.1% cottonseed meal, and 16.1% canola meal. Diets were equivalent, at 16.5% CP and 1.55 to 1.58 Mcal NE/kg DM. The DMI was decreased (P < 0.01) and was the lowest among diets for the urea treatment. This was not surprising because daily urea intake was 420 g, an excessive amount given the previous level of 135 to 136 g found not to limit intake. Correspondingly, milk production was lower (P < 0.01), milk fat percentage tended to be the highest (owing to both lower milk production and the rumen pH effect), milk urea N was higher (P < 0.01), and BW gain was lowest (P < 0.01) for the urea diet compared with the other diets. Most recently, Broderick and Reynal (2009) fed diets containing 40% (DM) corn silage and 15% alfalfa silage along with a concentrate primarily composed of shelled corn and soybean meal. The control diet and other diets contained 16% CP, with other diets having RDP increased by the addition of 0.41, 0.84, and 1.31% urea, whereas, correspondingly, dry shelled corn was partially substituted for rolled high-moisture corn and lignosulfonate-treated soybean meal was substituted for solvent-extracted soybean meal to modulate the increase in RDP with addition of urea. Daily urea intakes averaged 95, 193, and 292 g/cow per day for the 3 urea-containing rations. These early-lactation multiparous cows (12, averaging 66 DIM) and primiparous cows (16, averaging 119 DIM) were used in seven 4 × 4 Latin squares of 28-d periods, with the last 14 d for collection of intake and production data. Dry matter intake
decreased most (linear effect at \( P < 0.01 \)) for the diet with the greatest urea intake, with a similar pattern for milk production.

Cameron et al. (1991) used 4 midlactation, multiparous Holstein cows in a \( 4 \times 4 \) Latin square design. Diets were supplemented with urea (0.75%), starch, or both. Diets (on a DM basis) contained fishmeal with 35% alfalfa silage and 20% corn silage for the low-starch (34%) diets, whereas the high-starch (40%) diets had 35% alfalfa silage and 7.5% corn silage—9.4% starch and 3.1% dextrinose were added in place of lower corn silage. Intake, ruminal digestion, and passage to the duodenum of DM, OM, starch, ADF, and NDF were not affected by supplementing urea in the diets. Daily urea intakes were 175 and 158 g for urea and starch-urea diets. There was a tendency (\( P < 0.14 \)) for total DM digestibility to be increased by urea. Organic matter digested postruminally was increased (\( P < 0.04 \) or 0.07) by urea. This was primarily due to increased digestibility of ADF (\( P < 0.09 \)) and NDF (\( P < 0.11 \)) postruminally. Total VFA were increased (\( P < 0.10 \)) by urea supplementation. Rumen ammonia was increased (\( P < 0.0001 \)) with urea, whereas starch had a decreasing effect (\( P < 0.01 \)). Supplementing urea in diets increased N intake and percentage of dietary CP degraded in the rumen (\( P < 0.04 \)), but did not change the daily passage of total N, nonammonia N, and nonammonia nonmicrobial N. Urea increased microbial N passage to the small intestine by 40 g/d and improved the efficiency of microbial N flow to the small intestine, but these differences were not significant. Postruminal and total tract digestibility of N were increased (\( P < 0.05 \)) by urea supplementation. Intake of amino acids was not altered when cows were fed urea-containing diets, but passage of methionine to the small intestine was increased (\( P < 0.05 \)). Milk production was increased (\( P < 0.08 \)) by urea supplementation, whereas protein and fat concentrations were not altered. Although these data suggested that rumen ammonia was not limiting OM fermentation or microbial synthesis in the rumen, even though diets contained 3% fish meal, the increased fiber digestion in the large intestine indicated that the greater amount of rumen ammonia being recycled to the intestinal wall may have provided a source of N to enhance this postruminal fermentation.

**Rumen Ammonia Levels**

The proposition that rumen ammonia concentrations greater than 5 mg/100 mL are not beneficial to increasing rumen microbial production was first presented as an abstract (Satter and Slyter, 1972) and later published Satter and Slyter (1974). This was based on continuous in vitro fermenters using ruminal contents from steers fed either a protein-free purified diet, a corn-based all-concentrate diet, or a 23:77 forage:concentrate diet. A urea solution was infused continuously to supply a range of CP-equivalent diets. These results were the basis for the following concluding statement: “From this study it appears that once ammonia starts to accumulate in the rumen and exceed 50 mg NH\(_3\)-N/liter rumen fluid, nothing is gained by further application with non-protein-N” (Satter and Slyter, 1972). This became the basis for the landmark article mentioned earlier (Roffler and Satter, 1973).

Subsequently, ruminal samples were collected for 211 cows maintained under a variety of feeding programs (Roffler and Satter, 1975a). All rations in the 35 trials were formulated from natural protein sources. Crude protein and TDN were varied by changing ration ingredients and by altering their relative proportions. After such ration changes, cows were adapted to a ration for at least 1 wk before sampling. Cows were sampled at least 4 times daily. Most of the 1,033 rumen samples were collected via stomach tubing, with a few collected via rumen fistula. Mean ruminal ammonia concentrations were obtained by averaging results from the individual daily samples. The overall relationship between DM CP % and ruminal ammonia had an \( R^2 \) of 0.88. Dietary CP ranged from 8.1 to 24%, whereas TDN ranged from 53 to 84%. The authors noted that above 13% CP, ruminal ammonia rapidly increased beyond 5 mg/100 L and was in excess of what ruminal bacteria could convert to microbial protein. A model was developed to indicate, depending on both the TDN and CP of the diet without NPN, the theoretical upper limits for dietary NPN supplementation. Subsequently, ammonia saturation constants for representative pure cultures of predominant anaerobic and fermentative rumen bacteria were determined (Schaefer et al., 1980). For example, an organism with a saturation constant for ammonia of 50 mM growing in a medium containing 1 mM (i.e., 1.45 mg/100 mL) ammonia should achieve 95% of its maximum specific growth rate. However, these studies did not address what rumen ammonia concentration maximizes nonammonia-N flow through the abomasum or DM digestion in the rumen. Several studies addressing these aspects are summarized in Table 4. Allen and Miller (1976) fed sheep low-N, high-energy diets supplemented with graded levels of urea. Mehrez et al. (1977) fed sheep on whole barley with graded levels of urea by using automatic continuous feeders. Regular barley was the substrate used in the polyester bags. Pisulewski et al. (1981) also fed sheep continuously 1 of 3 diets, ranging from 12 to 50% barley straw, with inverse levels of barley, starch, and glucose. Five graded levels of urea were ruminally infused. The authors noted, “With the more normal practice of feeding twice daily, higher rumen ammonia concentrations might well be required to ensure that the requirement of microorganisms was always satisfied” Pisulewski et al. (1981). Erdman et al. (1986) fed dry, ruminally fistulated Holstein cows a 7.4% CP diet containing 50% cottonseed hulls, 47.4% corn, and 2.6% minerals and vitamins. The feedstuffs used as in situ substrates were corn meal, soybean meal, corn
gluten feed, cottonseed meal, and alfalfa hay. Treatments were continuous rumen infusion of 0, 73, 147, and 222 g urea/d using 3.5 L water as carrier. Rumen ammonia N increased linearly with urea infusion and resulted in mean values of 4.3, 10.1, 17.2, and 25.0 mg/100 mL. Higher rumen ammonia values occurred at or near feeding, with lower values at 2 to 6 h postfeeding. Maximum DM disappearance occurred at rumen ammonia N concentrations of 25 mg/100 mL for corn meal and soybean meal, and at 17 mg/100 mL for cottonseed meal and corn gluten feed. Only a slight effect of rumen ammonia N concentration was observed on alfalfa hay disappearance. When data were summarized from this study and from the literature in which rumen ammonia N and rumen or total-tract DM digestion were increased or estimated in vivo, increasing fermentability of feed increased the minimum rumen ammonia N, resulting in maximum DM digestibility values. However, considerable variation in this relationship resulted in an $R^2$ of 0.50.

**Dietary Fat and Urea Interaction**

A study by Hristov et al. (2004) evaluated the effect of a specific fatty acid (FA), lauric acid (LA; C12:0), on protozoa survival in the rumen. In the text, they briefly summarize a preliminary trial as follows: “In a preliminary trial, 4 levels of LA, 0, 0.2, 0.4, and 0.6% (as a percentage of an assumed rumen weight of 80 kg) were tested to determine the effect of LA on general cow health, DMI, rumen pH, and protozoal counts. Four cows were randomly assigned to 1 of the 4 treatments and treated twice (during the morning and afternoon feedings) for a period of 1 wk before measurements were taken. Following the first 2 treatments with LA, the cow on the 0.6% level went off feed, ruminal pH increased above normal, and protozoa were completely eradicated. Similar effects, although less severe, were observed for 0.4% LA. The cow subjected to 0.2% LA had a significantly reduced protozoal population, had a normal pH, did not show any obvious signs of discomfort, and did not decrease DMI.”

As a result of this preliminary trial and based on previous in vitro experiments (Hristov et al., 2000), they chose a level of 240 g/d per cow of LA (equivalent to 0.3% on an 80-kg rumen base) for this experiment. Treatments were control (water) and LA (240 g of LA/cow per day). Intake of LA was quite high because 240 g of LA/d per cow at a measured DMI of 23.1 kg/d equaled 1.04% LA of DMI. This contrasts with the preliminary trial, in which the authors found that 0.2% LA did significantly reduce protozoa, but cows had a normal rumen pH, did not show any obvious signs of discomfort, and did not decrease DMI. That level equates to 0.67% of DMI—still a very high level of DMI.

Previously, Newbold and Chamberlain (1988) used coconut oil and linseed oil for evaluations with sheep in vitro and in vivo, the latter with 2 different diets. Protozoal activity in vitro was measured as a percentage of the control for in vitro trials, and for in vivo trials, protozoal activity was measured in counts $\times 10^{-5}$/mL for the treatments (Table 5). Longer chain FA had a more deleterious effect on protozoa in vitro, and this was worst for the unsaturated long-chain FA C18:2. Coconut oil (rich in C12 and

### Table 4. Optimal rumen ammonia level from different references

<table>
<thead>
<tr>
<th>Reference</th>
<th>Method</th>
<th>Animal</th>
<th>Parameter</th>
<th>Optimal [ammonia], mg/100 mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allen et al. (1976)</td>
<td>In vivo</td>
<td>Sheep</td>
<td>Nonammonia-N flow</td>
<td>16 to 22</td>
</tr>
<tr>
<td>Mehrez et al. (1977)</td>
<td>In situ</td>
<td>Sheep</td>
<td>DM disappearance</td>
<td>&gt;23.5</td>
</tr>
<tr>
<td>Pisulweksi et al. (1981)</td>
<td>In vivo</td>
<td>Sheep</td>
<td>Microbial protein flow, $^{35}S$</td>
<td>2.3 to 8.7</td>
</tr>
<tr>
<td>Erdman et al. (1986)</td>
<td>In situ</td>
<td>Cows</td>
<td>DM disappearance</td>
<td>17 to 25</td>
</tr>
</tbody>
</table>

### Table 5. Coconut and linseed oil effect in vitro and in vivo on protozoa

<table>
<thead>
<tr>
<th>Item</th>
<th>Variable</th>
<th>Variable</th>
<th>Variable</th>
<th>Variable</th>
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</thead>
<tbody>
<tr>
<td>In vitro</td>
<td>C12:0</td>
<td>C18:0</td>
<td>C18:2</td>
<td></td>
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<tr>
<td>Percentage of control</td>
<td>71</td>
<td>55</td>
<td>14</td>
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</table>

<table>
<thead>
<tr>
<th>In vitro</th>
<th>Coconut oil</th>
<th>Linseed oil</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage of control</td>
<td>74</td>
<td>39</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>In vivo, mL/d (diet 1)</th>
<th>0</th>
<th>50</th>
<th>100</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coconut oil</td>
<td>5.1</td>
<td>2.5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Linseed oil</td>
<td>5.7</td>
<td>4.2</td>
<td>1.9</td>
<td>0.7</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>In vivo, mL/d (diet 2)</th>
<th>0</th>
<th>50</th>
<th>100</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coconut oil</td>
<td>2.1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Linseed oil</td>
<td>2.56</td>
<td>1.38</td>
<td>0.22</td>
<td>0</td>
</tr>
</tbody>
</table>

1From Newbold and Chamberlain (1988).
C_{18} saturated FA) was not as deleterious on protozoa in vitro as linseed oil (rich in C_{18} unsaturated FA). But in vivo, coconut oil appeared to have a more deleterious effect than linseed oil. In addition, linseed oil did not affect intake, but coconut oil at >50 mL/d reduced intake markedly. Considering the work of Hristov et al. (2004), it appeared that coconut oil, through its C_{18} FA content, had a more negative effect on DMI. Sutton et al. (1983) used 5 sheep in a 5 × 5 Latin square trial in which the sheep were fed a control diet or 40 g daily of each of the following: linseed oil, coconut oil, protected linseed oil, or protected coconut oil. Oils constituted 6.67% of DMI. Formaldehyde-treated sodium caseinate was used to protect the oils and was included in other diets too. Briefly, both free oils had similar effects on reducing OM and NDF digestion; coconut oil reduced ruminal protozoa by 90%, compared with a 78% reduction for linseed oil; and flows of total N and microbial N were increased by both oils, as was microbial protein synthesis (g N/kg OM apparently digestible in the rumen). This was due to reduced protozoal numbers allowing for more efficient microbial protein synthesis. Protected oils reduced these effects of unprotected oils, but because they were not entirely eliminated, this indicated that some degree of protection was lost in the rumen. They concluded that free oils could increase the efficiency of microbial protein synthesis, possibly by their defaunating effect, which may enhance the potential for using NPN with oil-supplemented diets.

When protozoa are reduced or eliminated, their role in engulfing starch and deaminating protein, thereby providing more rumen ammonia, is diminished or eliminated. In such situations, dietary urea could make several contributions, namely, minimizing the normal decrease in rumen pH through the hydrogen uptake step of urea hydrolysis after consumption, and providing for a more favorable rumen ammonia level.

**Urea and Nitrates**

Adams (1961) indicated that some persons relate nitrate poisoning with urea feeding, probably because nitrate can be chemically reduced to ammonia. Theoretically, nitrates could contribute to overloading the rumen with ammonia if urea were also greatly contributing to rumen ammonia levels. This is not likely for several reasons. The reduction of nitrate (NO\(_3^-\)) to nitrite (NO\(_2^-\)) is dependent on the presence of nitrate reductase. Normally, this enzyme is present and is not limiting in the rumen. Further reduction of nitrite is dependent on nitrite reductase, which is limiting and must be induced. Thus, if there is not time for this induction to occur because of rapid exposure to higher levels of dietary nitrates, nitrites produced in the rumen are rapidly absorbed into the blood. These nitrites convert blood hemoglobin to methemoglobin, which reduces oxygen transport to tissues, causing classic “nitrate poisoning” symptoms and even death. Thus, this poisoning is due to nitrites, not nitrates per se. Conversion of nitrates to ammonia is a slow process, whereas hydrolysis of urea to ammonia is rapid.

Several studies have addressed the urea-nitrate interaction and nitrate utilization. Addition of 2% potassium nitrate to lamb rations with or without 1% urea resulted in no differences in feed utilization and ADG (Carver and Pfander, 1974). The authors concluded that there was no toxicity effect from properly supplemented rations containing urea when up to 2% potassium nitrate was also included. In a second study, lambs were fed up to 4% potassium nitrate with 1 or 2% urea-containing rations with no sign of toxicity.

Murdock (1972) fed cows producing 22 to 27 kg milk daily green-chop forage with a DM nitrate content varying from 0.5 to 2.3%, and oat hay with a DM nitrate content varying from 1.6 to 4.0%, with no problems observed. Apparently, cows were able to adapt to the high nitrate levels and even used nitrate as an NPN source.

Problems usually occur with nitrates when they are at a high level in a diet and are introduced so quickly that cows cannot adapt quickly enough to avoid nitrate accumulation and nitrite toxicity. No firm data exist indicating that dietary urea is exacerbated by high dietary nitrates, resulting in ammonia toxicity.

**Urea and Reproduction**

Urea has been implicated anecdotally with decreased reproduction for years. A Michigan study (Ryder et al., 1972) summarized Dairy Herd Improvement Association records from 1965 to 1969. A total of 3,157 herd-year observations were collected, representing 85,281 calving intervals. Fifty-four percent of herds used urea. Of the total urea intake per cow for herds fed urea, 40 g came from urea-treated corn silage, 0.6 g came from urea-treated high-moisture corn, 7.5 g came from dry grain mixed on the farm, and 32.6 g came from commercial protein supplements (Table 6). The maximum amount of urea fed to any single herd was 370 g/cow daily. Correlations were done among 19 variables studied to relate calving interval with urea feeding. None was statistically significant. Multiple correlation coefficients indicated that only approximately 15% of the total variation in adjusted calving interval was accounted for by the 19 variables.

An extensive study was done with 81 Holstein heifers 7 to 12 mo of age, beginning in September 1968 (Erb et al., 1976). Heifers were fed diets in which urea replaced 0, 50, and 100% of the supplemental protein. Daily urea intake for the high-urea group averaged 16 g before breeding, 191 g during breeding and early gestation, and 68 g during midpregnancy. All rations were fed as TMR. Fourteen days before predicted calving, heifers were changed to lactation rations containing 0, 18, and 36% of the total N from 0, 0.9, or 1.8% urea. Rations contained 44% corn silage and 10% alfalfa bromegrass silage, and were fed once daily as TMR. During trial 1, average daily intake of urea was 0,
180, and 360 g at peak lactation. The authors reported that the highest producing cows in the high-urea group may have consumed in excess of 500 g urea daily in early lactation. Heifers calving during August to November 1971 were assigned to either a nonurea or a high urea-containing ration (trial 2) and were fed and managed as in trial 1. The only significant calving and reproductive traits are shown in Table 7.

Although retained placenta differed \( P < 0.05 \) between treatments in trial 2, rates were similar in the subsequent 2 calvings. Abortion rate was greater \( P < 0.05 \) for cows in the 1.8% urea treatment in trial 1. However, that rate was deceptive because the 14% represented 5 animals, 4 of which were heifers that had not entered their first lactation. Thus, only one abortion occurred in lactating cows in the 2 trials (C. H. Noller, Department of Animal Science, Purdue University, West Lafayette, IN, 1991, personal communication). The only difference in calving interval occurred in trial 2, in which the gestation length of cows in the nonurea treatment decreased in trial 2. There was also a significant 2- to 3-d decrease in gestation length for urea treatments in approximately one-fourth of the male-female pregnancies during the 4 pregnancies of the trials. However, this was primarily a reflection of little variation in gestation length and was of no real biological significance. Milk yield did not differ. The trend toward decreased milk yield with the higher urea level was because intake tended to be lower in early lactation. (The authors noted that cows on the 1.8% urea treatment ate more frequently and took longer to consume the feed offered at each feeding.) Milk production curves were thus altered, being lower in early lactation but higher in later lactation. Thus, the 200-d lactation yields were biased against the urea treatments. The 305-d yields were used because they were available to the authors; however, the organizer and fund procurer for this project (C. H. Noller, Department of Animal Science, Purdue University, 1991, personal communication) disagreed with this use of 305 d rather than the actual milk production records, along with several other aspects, and removed his name from the publications.

Four years later, the senior and third author of this study published a chapter on Effects of Urea on Bovine Reproduction (Erb and Callahan, 1980). Recognizing the data on abortions in the above study, they wrote, “Abortions were not observed when high-urea rations were not fed until the end of first pregnancy rather than prior to the first pregnancy or during subsequent years. Currently, there is no evidence that dietary urea caused abortions in cows after the first pregnancy.” Other significant comments included the following: “Failure to find repeatable year-to-year differences in reproductive parameters may indicate variations due to other factors or prolonged adaptation to high levels of urea. However, there is no reason to believe that the limits recommended for efficient utilization of urea for maintenance, growth, and lactation of breeding age cattle would affect reproduction adversely; and based on the foregoing data, it appears unlikely that daily intakes of up to 227 g of urea in high energy rations (daily intake of 22.7 kg DM containing 1% urea) would affect reproduction in lactating dairy cows.”

<table>
<thead>
<tr>
<th>Item</th>
<th>Trial 1, % urea</th>
<th>Trial 2, % urea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retained placenta, %</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Abortion, %</td>
<td>0( ^a )</td>
<td>0( ^a )</td>
</tr>
<tr>
<td>Calving interval, d</td>
<td>420</td>
<td>409</td>
</tr>
<tr>
<td>FCM, kg</td>
<td>3,464</td>
<td>3,433</td>
</tr>
</tbody>
</table>

\( ^a \)Means with different superscripts within a row and within a trial differ \( P < 0.05 \).

\( ^b \)From Erb et al. (1976).

\( ^c \)200-d lactation yields.
Synchronizing Urea Release

Johnson (1976) summarized the 3 categories of dietary carbohydrates (CHO) as 1) cell wall CHO, such as cellulose and hemicellulose; 2) readily fermented forms of glucose polymers, such as starch and dextrins; and 3) various forms of simple sugars, such as those that exist in molasses. He constructed a theoretical graph of their fermentative activity in rumen fermentation, which illustrated that cellulose is digested slowly over a long time, whereas sugars are digested over a shorter time, with starch in between these 2 CHO categories. Consequently, fermentation of starch would favor the utilization of NPN over fibrous CHO and simple sugars, such as in molasses. Assuming that most rumen microorganisms utilize N from the ammonia pool in the rumen, then the ideal situation in the rumen would be for the ammonia pool to have a curve similar to the CHO fermentation curves. However, the ammonia release patterns from most NPN sources do not conform to the pattern shown for source X, and the author could not visualize NPN sources that conform to the needs of all 3 types of CHO. Although most rumen microorganisms can utilize ammonia to a high degree, some may require amino acids or peptides. Thus, Johnson (1976) could not visualize a way “for NPN sources to conform to the needs for all three types of CHO, assuming the animals are fed only once or twice daily and that the groups of microorganisms which utilize the three classes of CHO all have the same ability or requirement to utilize N which is passed through the ammonia pool as the major source of nitrogen.” He also discussed some enigmas, such as molasses increasing ruminal fermentation but apparently not increasing microbial protein synthesis, and the scenario whereby with starch, “cellulose digestion was depressed when urea was utilized as a source of nitrogen. On the other hand, in the absence of starch considerably more cellulose was digested. Although this could represent competition for a number of nutrients besides nitrogen, some of this competition could be alleviated by simply adding additional nitrogen as urea to the system.”

In an extensive review, Reynolds and Kristensen (2008) found little evidence of benefits to synchronizing N with CHO. They attributed this to several factors: 1) recycling via blood and gut exchanges of urea and ammonia, a multitude of factors controlling urea transfer to the gut from the blood, and the inherent adaptability of rumen microbes to asynchronous N and energy supply. Although many of the studies reviewed were done with beef cattle and sheep, a significant number of dairy cattle studies were also included. They concluded that the intuitive benefits of rumen asynchrony in terms of the efficiency of N utilization have not typically been observed in practice.

Commercial Urea Products

Starea was “an intimate mixture of gelatinized starch and urea” patented by the Kansas State University Research Foundation to improve the utilization of urea N and palatability of urea-containing rations (Helmer et al., 1970a). It was produced as follows: “Starea supplement (23% CP) was a mixture of finely ground corn and urea processed in a Wenger X-50 Continuous Extruder Cooker, as previously described (Helmer et al., 1970b). The extruded material was passed through a hot-air dryer to reduce moisture content to 10 to 11%. The dried material was further processed through a corn cutter, crumble rolls, and a rotary sieve to produce particles the size of cracked sorghum grain.” In a 3 × 3 Latin square trial (Helmer et al. 1970a) with 6 cows/treatment fed brome-alfalfa hay and concentrate twice daily in a stanchion barn, cows consumed 7.6, 12.8, and 14.4 kg concentrate daily and produced 14.4, 17.6, and 18.3 kg milk daily. Consumption of concentrate and milk produced was decreased ($P < 0.05$) for the urea versus the Starea and soybean meal concentrate diets, respectively. However, urea content was 2.1% in the Starea-containing diet and was 2.8% in the urea-containing diet, resulting in 269 and 213 g of daily urea intake, respectively. Although cows consumed more urea with Starea, there were numerical declines in both intake and milk production for Starea compared with soybean meal. However, these levels of urea intake still exceeded by far the 135-g level of unprocessed urea found not to be limiting intake (Kertz et al., 1982). In a previous study, Helmer et al. (1970b) did find that Starea reduced ($P < 0.05$) rumen ammonia levels in vitro compared with unprocessed corn and urea, and this was associated with increased ($P < 0.05$) bacterial synthesis. Helmer and Bartley (1971) extensively reviewed studies evaluating ammonium salts, amino acids, amides, and amidines as well as urea derivatives and coated urea (CU). Although the ammonium salts were equivalent to urea at all levels tested, they had an inherent disadvantage because of their lower N content. This generally makes them, and many other NPN sources, more costly than urea on an equivalent protein comparison. Amides and amidines were generally not always hydrolyzable. The urea derivatives tested (biuret, n-butylurea, and others) inhibited bacterial growth and were not extensively hydrolyzed. Of these, biuret has been evaluated the most extensively (Huber and Kung, 1981). Its greatest limitation is that it requires a long time for rumen bacteria to adapt to it for use as an NPN source (and there is not unanimity as to this length of time), and then that adaptation is rapidly lost when the feeding of biuret ceases. It is also somewhat puzzling that production studies as well as N balance studies have indicated that biuret is utilized somewhat better by sheep than by cattle.

Coated urea or slow-release urea (SU) products have been developed to slow ammonia release, reduce toxicity, provide more efficient utilization of N, and improve palatability. Helmer and Bartley (1971) acknowledged that CU products had not gained commercial acceptance. They at-
tributed this partial slowing down of conversion of urea to ammonia as only one factor in its utilization. Because the majority of rumen bacteria prefer ammonia to amino acids, they postulated that it may be better to permit production of ammonia from urea but to encourage the more efficient utilization of ammonia. Additional processing also increases the cost and reduces the protein equivalent level compared with unprocessed urea.

Researchers in Ohio developed Dehy-100, a pelleted mixture of 32% urea alfalfa meal, dicalcium phosphate, sodium sulfate, and sodium propionate that contained 100% CP equivalent with approximately 3% urea (Conrad and Hibbs, 1968; Huber and Kung, 1981). Although milk production was lower for cows fed Dehy-100 in both a research herd trial and a local field trial, greater milk fat percentages resulted in similar 4% FCM production, although intake of the concentrate was noticeably less. This likely reflected the fact that at the maximum intake, 454 g of urea and 908 g of dehydrated alfalfa were being consumed. Both of these components would contribute to greater milk fat percentages. Urea would have contributed approximately 40% of total CP intake.

A polymer-CU product (Optigen 1200 Controlled Release Nitrogen, CPG Nutrients Inc., Syracuse, NY) was used at 3 levels (0, 0.77, and 0.77%), whereas unprotected urea was 0.30, 0.13, and 0.09%, respectively, to determine dairy production responses (Galo et al., 2003). Diets fed as TMR contained approximately 28% corn silage, 15.7% grass-legume haylage, 6.8% chopped western alfalfa hay, 24 to 29% ground corn, and other ingredients, including 0.76 to 1.15% Ca salts of FA. Diets were formulated to be isoenergetic at 1.76 Mcal NE/kg DM. The CU contained 97% urea and 3% coating by weight. There were several limitations to this study: no negative control; different levels of Ca salts of FA, which affect DMI (NRC, 2001); differing levels of fish and blood meal, which affect DMI; no indication of how urea:CU levels were determined; and in vitro evaluations indicating that the release rate of CU was more rapid than expected because of mechanical damage of the coating. The only significant differences were lower \( (P < 0.05) \) milk production with the 16% CP + CU diet. Urea:CU daily intakes per diet were 71:0, 31:182, and 21:178 g, respectively. The threshold of 135 g urea/cow daily was exceeded with an SU product that was not truly protected.

Golombeski et al. (2006) used an SU product in concert with highly fermentable sugars (FS) and measured production responses in Brown Swiss cows. The FS was Rationmate (Midwest Ag Enterprises, Marshall, MN) and consisted of whey permeate and corn steep liquor blended into a single liquid feed product. The composition was 48.5% DM, and as percentage of DM, the composition was 22% CP, 2.54% starch, 24.2% total sugars, 23.5% lactose, 0.74% Ca, 2.09% P, 3.84% K, 0.57% Mg, 1.32% S, 1.98% Na, and 2.70% Cl. The SU was Ruma Pro (XF Enterprises, Hereford, TX), a liquid CaCl₂-bound urea with slow-release properties. There was no indication of how the formulated SU levels were determined or what percentage of the SU product was urea. Diets fed as TMR contained 15% alfalfa hay and 35% corn silage, and varied in FS and SU, with a range of 15.7 to 16.5% CP and a formulated level of 1.55 Mcal NE/kg DM. The DMI was lower \( (P < 0.01) \) for SU versus no SU, and daily intake per cow of SU without FS was 120 g and was 122 g for SU with FS. The authors speculated that DMI was less with the SU diets in relation to the “bitter taste of urea suggested by Huber and Kung (1981).” They also stated that “the mechanism of intake depression is not completely understood,” indicating their lack of familiarity with the literature covered in the current review. They also cited findings by Casper and Schingoethe (1986) of lower DMI with urea-containing rations. In that study, cows were fed 50% concentrate rations that contained no urea, 1% urea, or 1% urea + 30% dried whey. The DMI of these TMR diets were 22.0, 20.2, and 23.1 kg/cow daily, respectively, and differed \( (P < 0.05) \) among each other. Cows were housed in a freestall barn, fed TMR with Calan gates, and monitored 3 d for 24-h periods for eating time. There were no differences among diets in total minutes of eating time per cow daily, but cows on each diet differed \( (P < 0.05) \) among diets for meals per day and for minutes per meal. Going from the control diet through the 1% urea and 1% urea + whey diets, cows increased \( (P < 0.05) \) the number of meals per day while at the same time decreasing \( (P < 0.05) \) the minutes per meal, resulting in the same total minutes of eating per day. This was surprising because daily urea intakes averaged only 101 and 116 g/cow daily for the latter 2 diets. However, there was no indication of the level of CaCl₂ in the SU product, and CaCl₂ can be an intake depressant for cows, depending on its dietary level. Milk production approached \( (P < 0.06) \) being lower for the FS diets, whereas milk fat percentage was higher \( (P < 0.002) \) for the FS diets. The latter is consistent with whey-fed diets resulting in greater milk fat percentage. However, in this trial, ruminal butyrate was higher \( (P < 0.001) \) for the SU diet compared with the FS diet, whereas ruminal acetate:propionate was higher \( (P < 0.07) \) for the FS diet compared with the SU diet.

### Urea and Added NPN Sources in the Total Diet

Huber and Kung (1981) reviewed NPN sources and their utilization, particularly with NPN added to silages, especially corn silage. A further dimension evaluated was how this also related to including urea in the concentrate (Shirley et al., 1972; Huber et al., 1980a). It has been shown (Huber et al., 1979, 1980b) that 40% of the ammonia in ammonia-treated corn silage was converted to protein during the fermentation process and that ammonia prevented the degradation of corn silage protein by approximately 20% compared with untreated corn silage. Thus, ammonia-treated
corn silage is not necessarily greater in NPN than in regular corn silage. Huber et al. (1979) found that ammonia treatment of corn silage provided approximately 20% of CP from NPN. Addition of urea to the concentrate provided another 5.2 to 10.7 percentage units of CP from NPN. There were no negative effects on intake or milk production in these trials. The addition of urea to corn silage produced similar results. Shirley et al. (1972) found that CP from the urea treatment of corn silage provided 14 to 26% of total CP. Milk production did not differ. In all these studies, corn silage provided 60 to 74% of total DMI. Four experiments were done over 4 consecutive years (Huber et al., 1980a) with 96 cows fed an average of 55% of DMI from corn silage. There were no negative effects on DMI and milk production when 20% of total CP came from NPN treatment of corn silage, and urea in the concentrate added another 13% of CP from NPN. Thus, it would be conservative to allow no more than 20% of total CP coming from all added dietary NPN sources without any negative effects. These data indicate that up to 30% could come from all added dietary NPN sources, depending on other conditions.

**Toxicity and Urea Feeding Levels**

As indicated previously in the section Intake Depression Mechanism, Bartley et al. (1976) found rumen pH to be most highly correlated with ammonia toxicity, rather than with rumen ammonia levels. That is because rapid hydrolysis of large amounts of urea in itself increases rumen pH, which then allows more rapid absorption into the blood of now higher levels of rumen ammonium. Huber and Kung (1981) found that large amounts of urea (more than 45 to 50 g/100 kg BW) in a short period of time can be fatal to unadapted cattle. Bartley et al. (1976) observed muscle tetany at approximately 53 min after a dose of 50 g urea/100 kg BW delivered through a rumen cannula. The classic treatment for ammonia-urea toxicity (Huber and Kung, 1981) was “orally drenching animals with acetic acid (5 to 10% solution) as soon as urea toxicity was observed. This was followed by a second drenching 2 to 3 h later, with 50% of the initial dose effectively preventing death of cattle challenged with toxic amounts of urea in Oklahoma studies (Word et al., 1969). Treatment with acetic acid concentrations stronger than 10% are not advised because of injury to the esophagus. In the Kansas State study (Bartley et al., 1976), drenching the rumen with acetic acid after toxic amounts of urea were administered did not lower rumen ammonia in 10 min, and 10% of the treated animals succumbed. Rapid evacuation of rumen contents was totally effective for preventing death from urea toxicity.”

**Implications**

The traditional urea feeding recommendations have been that urea not contribute more than one-third of total dietary CP, not more than 3% of concentrate, and not more than 1% of the total ration. The first recommendation even exceeds the recommendation of 25% of total CP coming from urea (Reid, 1953). For a cow consuming 22.7 kg of a 16% CP TMR with 50% forage, the above recommendations would correspond to 427, 341, and 227 g daily urea intake, respectively. In many instances, we know these amounts would be excessive, especially when eating time is limited or TMR are not being fed. The most obvious problem would be reduced intake via the conditioned negative aversion response. These amounts would also be excessive using the metabolizable protein method of ration balancing (NRC, 2001). Based on this review, a more reasonable recommendation (Kertz, 1982) for feeding urea to dairy cows is 1% of the concentrate, 135 g/animal daily, and not more than 20% of total CP, counting other added NPN sources. These feeding levels would be appropriate even under the most adverse conditions for maintaining normal DMI. Depending on other conditions, the urea source, and ration balancing programs, these levels may be safely exceeded.

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**LITERATURE CITED**


Urea feeding to dairy cattle


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