Non-protein Nitrogen in the Feeding of Cattle

Allen D. Tillman, *Ph.D.*
Department of Animal Sciences and Industry
Oklahoma State University
Stillwater, Oklahoma 74074

Protein is the first-limiting nutrient in cattle production worldwide. The deficiency is more acute in tropics because of the long and variable dry seasons and the reseeding difficulties of legumes. Urea and other non-protein nitrogen (NPN) sources can be used to supply some of the needed dietary nitrogen for all ruminants. In recent years, greater usage of the cereal grains in cattle rations has also improved ration conditions for use of NPN compounds by cattle. The purpose of this paper is to review some factors affecting NPN utilization by cattle and to give some guidelines for using these compounds in their feed.

Mechanism of NPN Utilization

In 1879, Weiske, et al. (1), reported that ruminants, because of microbial action in the rumen, were able to use NPN compounds as a partial substitute for dietary protein. Since that time, many researchers have studied the mechanism of NPN utilization in the rumen of cattle. Their results support the idea that ammonia is the common denominator in the utilization of any NPN compound (2) and, if the nitrogen-containing compounds cannot be hydrolyzed to ammonia, they have no value as a protein substitute for ruminants.

If urea is the substrate, the following steps appear to be involved in its complete conversion to bacterial protein:

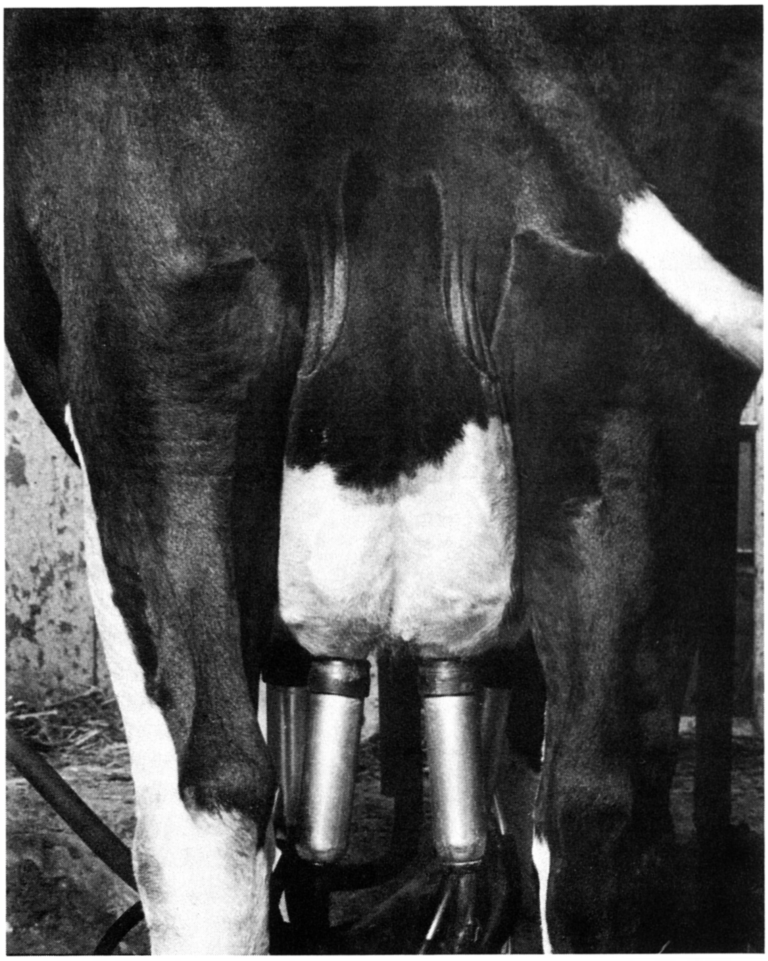
1. Urea $\xrightarrow[\text{Urease}]{\text{Microorganism}}$ $NH_3 + CO_2$
2. Carbohydrates $\xrightarrow[\text{Enzymes}]{\text{Microorganism}}$ Volatile Fatty Acids (VFA) + Keto Acids
3. NH_3 + Keto Acids $\xrightarrow[\text{Enzymes}]{\text{Microorganism}}$ Amino Acids
4. Amino Acids $\xrightarrow[\text{Enzymes}]{\text{Microorganism}}$ Microroganism proteins
5. Microorganism Proteins $\xrightarrow[\text{\& Small Intestines}]{\text{Animal Enzymes in the Abomasum}}$ Amino Acids
6. Free amino acids are absorbed from the small intestines and used by the animal.

A similar scheme can be put forth for each NPN source that requires enzymic hydrolysis to ammonia.

In the case of urea, step 1 is about four times as fast as step 2, even when the carbohydrate source is readily fermentable (3). When poor-quality roughage sources are used as the energy source, differences between steps 1 and 2 are wider. If the keto acids are not present in rumen fluid at the time needed, the liberated ammonia is absorbed across the rumen wall into the blood and the body has to expend energy for the conversion in the liver of blood ammonia to urea. More energy has to be expended later in the filtration by the kidneys of this urea from the blood and into the urine. The net result of both of these steps is to reduce the efficiencies of the utilization of dietary energy and protein. For efficient ammonia utilization, there is need for a system in which the rates of both ammonia and keto acids production are so co-ordinated that both would appear in the rumen fluid as needed for amino acid synthesis (4). The practical nutritionist needs to be guided by fundamental studies on the kenetics of NPN utilization and could use the results of such studies as a basis for designing practical experiments for improved NPN utilization (5). In this connection there is currently much interest in biuret as well as in mixtures of biuret and urea to be used for supplementing rations composed primarily of low-quality forages, which contain high levels of the lignocellulose complex.

Many rumen microbes prefer ammonia nitrogen to peptide nitrogen for the synthesis of their body proteins (2). Since dietary proteins are hydrolyzed to amino acids, which can be deaminated, the needs of these ammonia-prefering bacteria can be met by dietary protein (6), but urea or other NPN sources could supply this need.

(for treatment)



BOVAMYCIN* II
Mastitis Treatment

A highly effective combination of ingredients for treatment of mastitis in the lactating cow. Micro-homogenized drug particles are suspended in a functionally designed base, allowing the rapid release and thorough dispersion of active ingredients throughout diseased tissue. Each dose is supplied in a convenient, disposable syringe, including a bacteria-free syringe tip and a PREPTIC** Swab for cleansing the teat orifice—all ready to use.

BOVAMYCIN II Mastitis Treatment . . . The preferred treatment for rapidly returning the infected milk cow to productivity. Each dose contains: procaine penicillin G, 100,000 units; dihydrostreptomycin sulfate equivalent to dihydrostreptomycin base, 50 mg; hydrocortisone acetate, 20 mg.

WARNING: Milk that has been taken from animals during treatment and for 60 HOURS (5 milkings) after the latest treatment must not be used for food. Administration of more than 2 doses in any quarter may result in drug residues in milk.

*Trademark
**Trademark of Johnson & Johnson

The ruminant animal, because of the action of rumen microbes upon protein, is wasteful in his utilization of dietary protein. In other words, no matter what the source or quality of dietary protein, the microbes will hydrolyze it to amino acids and then further degrade some of these to ammonia and a keto acid, and then build completely new protein systems from the nitrogen present in rumen fluid.

Many studies have shown that microbial protein contains a high level of nucleic acid, which is poorly utilized by the ruminant animal (7,8). Also, microbial proteins are unbalanced as regards their contents of certain dietary essential amino acids. Therefore, the ruminants' growth rate may be inhibited by an unbalanced microbial protein even though the diet contained high-quality dietary protein (9). Some workers (10,11) have found that the treatment of dietary proteins with formaldehyde or the tannins can cause cross linkages in the proteins which reduce microbial degradation of the treated protein thereby effecting a "bypass" of that protein. The treated protein, however, will be digested by the animals' enzymes found in the abomasum and small intestines.

This opens up a whole new area for research using the following rationale: If a NPN compound could be added to supply just enough dietary nitrogen to provide for a vigorous microbial population needed for the maximum degradation of the ligno-cellulose complex to supply energy for the animal, and for the production of some poor-quality protein by this means, one could then supply a dietary protein which has a dietary amino pattern to supplement the deficiencies of the microbial protein, and treat it in such a manner so as to effectively bypass degradation in the rumen. It now appears possible that the use of such a system would make it possible to obtain maximum protein synthesis in animals consuming a high-roughage diet. The possibilities are numerous and this is an active field of research around the world.

Ration Factors Affecting Urea Utilization

Urea, from the standpoint of usage, is by far the most important NPN compound, having a current usage of about 680,000 tons per year (12). This amount of urea would replace the nitrogen equivalent of about 4.5 million tons of preformed protein supplements. Because of greater usage, more research has been directed toward the effective utilization of urea than any of the other NPN sources.

Reid (13), as early as 1953, reviewed the research work on urea and reported on many of the basic factors affecting urea utilization by ruminants and many of these hold at the present time.

Level and solubility of dietary protein: When urea is included in diets containing low levels of protein and high levels of readily fermentable carbohydrates it is well-utilized. Conversely, the inclusion of urea in diets containing high levels of dietary protein and low levels of readily fermentable carbohydrates results in poor utilization and could result in ammonia toxicity, especially if the dietary protein is readily soluble in the rumen fluid.

More recently, Burroughs, et al. (14,15,16), have proposed greater restrictions than used previously for the use of urea in beef cattle rations. Their proposal makes use of a new evaluation of feeds based upon their estimated urea fermentation potential (UFP) and an efficiency of urea nitrogen conversion of 40% when the ration has a positive UFP value. UFP values have been estimated on the basis of the readily fermentable energy of a given feed or ration and the amount of feed or ration protein degraded in the rumen when consumed by cattle (3). This system, which will be discussed in more detail later in this paper, will readily indicate when rations will or will not be benefited by urea supplementation.

Corn or maize, which contains a high level of readily fermentable carbohydrate and a low level of crude protein which is only slowly soluble in rumen fluid, is an excellent feed to be used with urea. The starches from other cereal grains also have high utility in urea utilization, depending in part upon their crude protein levels.

Dietary Carbohydrates: The source of keto acids for amino acid synthesis in the rumen is primarily dietary carbohydrates. Starches and sugars, which are readily fermentable, promote better urea utilization than cellulose or the ligno-cellulose complex. Researchers have found that the starches are superior to the simpler sugars for urea utilization. However, in spite of this, cane molasses is widely used as a carrier for urea to supplement the rations of cattle kept on dry range forages during the winter or dry seasons. Klett (17) has reviewed the use of liquid supplements in the United States and reported that economics in the feeding of the supplement is an over-riding factor in the selection of protein supplements. Less labor is required in feeding the liquid supplements, thus their usage is widespread. Satisfactory performance, even though lower than that found when urea-grain or preformed protein supplements are fed, is obtained in the liquid-fed cattle. Also, the liquid supplements permit the use of mechanical devices to limit rate and amount of intake, thereby reducing cost and the chances of ammonia toxicity in cattle.

Dietary Fat: Many workers have found that the simultaneous addition of dietary urea and fat to cattle ration result in reduced urea utilization. Fat is less efficiently used as a source of the keto acids needed for protein synthesis, thus it is necessary to meet the carbohydrate needs before fat is added, and if the carbohydrate levels are adequate, low levels of dietary fat can be used in cattle rations containing urea.

Minerals: Substitution of urea plus grain or molasses for preformed protein supplements can sharply reduce the mineral supply in the urea-containing ration and this important point is often overlooked by practical nutritionists. The presence of urea does not alter mineral requirements, however, the mineral additives could have different availabilities than those found in natural protein diets. For protection one must supply dietary levels of all minerals in compliance with the requirements for and availabilities of each essential mineral.

Adaptation Period: Many workers have found that a period of two to three weeks is required for ruminants to become "adapted" to urea feeding. During the adaptation period, feed intake and production rate is decreased. However, once the animal is adapted there is some compensatory effect upon growth or production, thus total production is not affected adversely. There is also evidence that the adaptation is to the ration rather than to urea, thus minor ration changes are indicated when urea is used to replace natural protein supplements.

Rate and Frequency of Feeding: Many workers have found that frequent feedings stimulate urea utilization over that found with less feedings, presumably because more frequent feedings avoided the peaks in ruminal ammonia levels found when once daily or every-other-day feeding is used. Frequent feedings give improvement in animal performance when the ligno-cellulose complex is relied upon to furnish the major source of keto acids needed for amino acid synthesis in the rumen. Under some ration conditions, frequent feedings do not improve urea utilization.

Dietary Antibiotics: A review of the literature on this point reveals that low levels of antibiotics in the diets of cattle appear to stimulate performance. However, the number of experiments are limited and there is need for further detailed studies to clarify this point.

Diethylstilbesterol (DES): The work of Karr (18,19) and others indicate that DES improved the utilization of urea-containing diets to a much greater extent than was obtained when preformed protein-containing diets were fed. Therefore, prohibiting the use of DES in rations of ruminants by the Food and Drug Administration appears to affect the utilization of urea at a time when there is a limited supply of preformed protein available for ruminant feeding.

Effect of Dietary Urea on the Utilization of Other Nutrients

There are many reports in the literature on results of experiments which were designed to study possible interactions between urea and other nutrients. In general, urea has not been found to affect adversely the utilization of other nutrients when it is fed at recommended levels in the diet. As was indicated earlier in this report, there are many research results which support the idea that certain added nutrients will improve urea utilization. However, these results can be explained, for the most part, on the basis of the supplying of limiting nutrients.

There appears to be no consistent adverse effect of dietary urea upon the utilization of the dietary carotenes or Vitamin A, even though some earlier experiments indicated some effect. Also, some workers have suggested that dietary urea might aggravate ration conditions in which the forages consumed by ruminants contain high levels of nitrates. In general, two ideas persist: some feel that dietary urea might increase the forage nitrate content. As dietary urea does not appear to be a precursor for nitrate production in rumen fluid, this idea does not have much merit. The other idea concerns the degradation of nitrates in the rumen to ammonia (23): If dietary urea were present, ruminal ammonia levels would be high, thus there might not be a need for nitrate degradation beyond the nitrate or nitrate stage. This is still an open question, and it is of interest to note that Smith and Hatfield (24) found that dietary urea tended to counteract the detrimental effects of dietary nitrates.

There is evidence that excess levels of rumen ammonia along with other factors present in certain forages will lessen the utilization of dietary magnesium. However, the proper usage of urea would not lead to abnormal ruminal ammonia levels. Also, recent results (34) indicate that even the high levels of ruminal and blood ammonia found in urea toxicosis did not reduce blood magnesium levels.

Practical Use of Urea in Cattle Diets

1. *Beef Cattle*

Based upon 46 feeds assigned UFP values, Burroughs, et al. (14,15,16), have made generalizations as to which feeds or rations will or will not be benefited by urea supplementation. In these generalizations it is possible to predict the usefulness of supplemental urea as compared to supplemental preformed protein. These generalizations are:

a. "Submaintenance to low-productivity cattle rations containing principally roughages or forages, such as weathered pasture grasses, cereal straws, cactus plants, corncobs, corn stover, cottonseed hulls, or soybean straw, containing less than 6 or 7% protein on a dry matter basis, will be benefited by proper urea supplementation, whether or not the supplement contains a high-fermentable-energy feed with a high UFP value. Additional benefits from proper urea supplementation will occur with each of these low-productivity cattle rations if the supplement also contains a high-energy feed with a high UFP value, such as cane molasses, corn grain, or milo grain. High-energy feed supplements with no UFP value, such as beet molasses, barley, or wheat grain, would not be expected to enhance these low-productivity rations from the standpoint of urea utilization, even though the energy and preformed protein they supplied would in themselves greatly benefit such rations. The low-productivity rations having total digestible nutrient (TDN) values less than 60% on a dry matter basis and supplying more than 6 to 7% protein would not be expected to be benefited by any amount of urea supplementation."

b. "Intermediate-productivity cattle rations with moderate amounts of fermentable energy in which the principal feeds supply 60 to 75% TDN of the dry matter will be benefited by urea supplementation if the protein content of the ration is less than 9 to 10%. Such rations will be benefited additionally by urea if the supplements contain a high UFP feed, such as cane molasses. In this instance, urea supplemental benefits would be expected with ration protein levels up to 10 or 11% of ration dry matter. On the contrary, these intermediate-productivity rations would not be expected to be benefited by urea supplementation if the ration protein exceeded 9 to 10% when no feeds in the supplement had a positive UFP value."

c. "High-productivity cattle finishing or lactation rations with more than 75% TDN in the dry matter will be benefited by urea supplementation if the protein content of the ration dry matter is less than 11 to 14%. No cattle ration with any amount of urea supplementation would be expected to be benefited if it contained more than 14% protein, irrespective of how much fermentable energy it supplied or the purpose for which the cattle were being fed."

In an evaluation of over 100 experiments in which urea was added, Burroughs, et al. (16), found the experimental results supported the above generalizations. When other parameters such as NFE:urea ratios (20) or the ratio of digestible energy to nitrogen (21) are considered, the conclusions of Burroughs, et al. (16), also appear to be valid. For example, Pigden (21) made the following recommendations:

a. When forages have no more than 50% digestible energy, 1% dietary nitrogen (6.25% crude protein) is adequate for its utilization.

b. When the digestible energy of the primarily roughage diet is higher than 50%, the N requirement is increased to 1.5%.

c. When readily available energy is added as starch at levels in excess of 20% of the ration, the N requirement increases to about 2%.

Burroughs (16) has now developed a feeding system based upon UFP of feeds. It is a logical system and offers promise of widespread usage.

2. *Dairy Cattle*

Helmer and Bartley in 1971 (22) and Coppock (personal communication) reviewed much of the recent research work on the feeding of urea to dairy cattle. One of the most important factors is the level to be fed to each production group of dairy cattle. However, there are many variables affecting levels of urea to be fed to dairy cattle, thus the guidelines set forth here include ranges which reflect many possibilities:

a. Urea can be included in starter rations for calves at levels from 1.25 to 2.00% of the total ration. Such rations must contain in addition to urea some high-quality protein such as soybean meal.

b. Growing heifer rations may contain 1.0 to 1.5% urea in the total ration.

c. Lactating dairy cows vary widely in the level of milk production. It appears that the high-producing cow cannot tolerate very high levels of urea and that the upper level is *0.45 kg of urea per 1000 kg of body weight.*

Another of the major factors in the use of urea in rations of high-producing cows is the problem of palatability or acceptability of urea-containing rations. Present research (22) results support the idea that acceptance is favored by the following factors:

a. Use of multi-ingredient concentrate mixtures which include industrial by-products such as molasses, distillers grains and other products.

b. Feeding systems which permit the cow to eat small amounts throughout the day.

c. Good mixing of the urea into the other ration ingredients, and,

d. The inclusion of urea into complete ration—diluting the effects of urea.

Newer products such as Dehy-100 and Starea appear to offer promise for improved utilization of rations by dairy cows.

Effect of Dietary Urea on Herd Health

Because of potential toxicity of dietary urea, some have implicated it in reproductive disorders, mastitis, milk fever, and many other disorders found in both beef and dairy cows. Rys (25) reported that urea has been routinely included at a level of 2% of concentrate mixtures for cattle for years in Poland and that there have been no symptoms of liver or kidney damage except in cases of acute poisoning caused by excessive intakes of urea.

However, there have not been many longtime and controlled studies on cattle receiving urea continuously as a dietary ingredient. Archibald (26) included urea at a level of 3% of the concentrate mixture for dairy cows in one long-time study and found that herd health and reproductive performances were not affected. In a shorter experiment of seven months, Colenbrander, et al. (27), found that dietary urea neither affected carcass quality nor the status of the organs or tissues, Patton, et al. (28), found that dietary urea did not affect daily gains, blood urea, services per conception and cycle lengths of repeat breeders when it was fed to Holstein cows; plant protein supplements were fed to the control animals.

Virtanen (29) and Oltjen (30) fed "purified" diets in which urea was the sole nitrogen source to dairy and beef cattle and found that those cows required more services per conception than comparable ones fed practical diets. As purified diets are so different from practical diets in many ways other than nitrogen source, it is doubtful that urea should be implicated.

Mistakes in mixing and other factors can result in sickness and death in cattle, urea toxicity. Many workers have made detailed studies on symptoms of urea toxicity, predisposing factors leading to toxicity, levels of urea necessary to cause toxicity, and method of treatment of affected animals.

As urea toxicity is ammonia toxicity, a discussion of ammonia is indicated. Ammonia is a weak base with a PKa of about 8.8 at 40°C, therefore, there is a close relationship between ruminal fluid pH and the level of free ammonia present. As indicated earlier, the action of urease in breaking down urea in the rumen is rapid. Also, there are many types of ruminal bacteria which produce urease, thus there is sufficient urease present to hydrolyze urea at a rapid rate under many ration conditions. Thus the feeding of high levels of urea results in a rapid release of ammonia into the rumen fluid. This condition will cause a rise in rumen fluid pH; buffering capacity against alkaline buildup in the rumen is not as pronounced as it is against an acid buildup. As the lipid layer of the rumen mucosa is permeable to ammonia, there is rapid absorption of the additional ammonia made free by the rise in ruminal pH. Many workers (31,32) have shown that increases in portal blood ammonia levels parallel increases in ruminal fluid ammonia concentrations. When the rate of ammonia absorption exceeds the rate at which the liver can convert ammonia to urea, there is an increase in total blood ammonia levels and toxicity may result if the level is high. *It appears that rumen fluid NH_3 - N levels of 80 mg/100 ml or above will cause toxicity and could be used as a diagnostic guideline.* Blood NH_3 - N levels causing toxicity symptoms are difficult to determine and it is suggested that levels *above 1.5 mg/100 ml of blood would be toxic.* Buck (33) has reported that in most cases of urea poisoning ruminal fluid NH_3 - N levels will exceed 100 mg/100 ml while blood levels will exceed 2 mg/100 ml. In using these levels as diagnostic tools, one must keep in mind that postmortem samples of blood and rumen fluid must be taken as soon after death as possible because proteolysis proceeds rapidly after death and could cause higher NH_3 - N levels.

The clinical course of urea toxicosis is rapid and is usually acute with death occurring from about 30 minutes to 240 minutes after consumption. The animal behaves as if it were experiencing abdominal pain and there is frothy salivation, grinding of the teeth and kicking at the abdomen. Polyuria, muscle tremors, weakness, incoordination, rapid breathing, violent struggling, bellowing and terminal tetanic spasms are also noted. The jugular pulse is marked and bloat occurs in most animals. There are usually no characteristic lesions found on necropsy examination; however, congestion, hemorrhages and pulmonary edema are common.

Predisposing factors to urea toxicity in cattle appear to be (1) lack of previous exposure to urea-containing diets suggesting that extreme care should be exercised when the animals are first exposed to urea-containing diets, (2) fasting, (3) high roughage diets, (4) the feeding of diets causing a high ruminal pH, and (5) low water intake. Circumstances which usually cause urea poisoning include (1) improper mixing of urea; (2) feeding urea to unaccustomed cattle; and (3) using high levels of urea in diets low in readily fermentable carbohydrates but high in crude fiber.

The best treatment for urea toxicity, if the cattle are found before terminal tetanic spasms are found, is to administer, and immediately, several gallons of cold water orally; five to ten gallons can be given. If a 5% acetic acid solution or vinegar is available, one gallon of either should be given along with the cold water. The cold water will lower the ruminal fluid temperature and thereby reduce the rate of ureolysis. It will also dilute the level of ammonia in the rumen and the rate of ammonia absorption from the rumen. Acetic acid will react

with ammonia, producing a neutral compound, ammonium acetate.

Misconceptions

Buck (33) and Floyd (34) point out that there are some common misconceptions among veterinarians and livestockmen with regard to the use of urea:

One misconception is that urea toxicity causes alkalosis in the blood and tissues. The work of Floyd (34) shows that the pH of blood drops from 7.4 to 7.0 at the time of urea-induced death. However, rumen fluid in toxic cases is alkaline with a pH range from 8.0 to 8.5.

There is also a misconception that some of the ammonia, which is liberated from urea by the action of urease, is eructated by the animal and aspirated into the respiratory tract, thereby causing irritation and increased susceptibility to respiratory infections. The facts are that, under the usual conditions of urea feeding, over 99% of the released ammonia is in the form of NH_4OH which is not gaseous. The small amount of ammonia present is soluble in the liquids of rumen fluid, thus it is also nongaseous. Therefore, unless the ruminal fluid pH was elevated above 8.0, no gaseous NH_3 would be available for eructation and under these conditions acute toxicity is common.

A third misconception is that urea is a chronic poison for cattle. This is not likely since the microflora can use the ammonia liberated from urea to synthesize protein. Many research projects have been designed to study the possibilities of producing chronic conditions in cattle and chronic effects *have not* been observed. Authorities state that, if urea toxicity occurs, it is acute and that death or recovery occurs within a few hours.

A fourth misconception is that urea feeding will cause reduced blood magnesium values. In Floyd's study (34) the increased blood ammonia levels, associated with urea toxicity, did not cause lowered blood magnesium levels.

A fifth misconception is that the incidence of abortions will increase in cow herds in which some cows have died as a result of urea toxicity. Ryley and Gartner (35) working with six pregnant cows given intraruminally 0.20 to 0.35 g urea/kg body weight at intervals of 104 to 237 days in the gestation period found that all surviving cows gave birth to healthy calves. In more extensive studies, the author's group (32) worked out a procedure, after the loss of 6 cows, whereby it was possible to drench pregnant cows, which ranged from 120 to 180 days post conception, with 0.44 g urea/kg body weight. The cows so treated showed definite symptoms of ammonia toxicity within 15 minutes and, if they were not treated, would die within 30-60 minutes. However, if they were given acetic acid 15 minutes after dosing with urea, they would appear to be normal for about the next 165 minutes and then the toxic symptoms would reappear causing death about 240 minutes after the urea was administered. However, only mild symptoms of urea toxicosis developed if a second dose of acetic acid was given 180 minutes after dosing with urea. Each treated cow was then paired with a control animal which was in the same stage of gestation and allowed to graze in a common pasture. There were no abortions in any animals, even though toxicity symptoms had been well advanced in many of the cows. The urea treatment did not affect birth or weaning weights of the calves, nor did it affect the rebreeding performance of the cows.

Use of Condensation Products

As was discussed earlier in this publication, urea is quite soluble in rumen fluid and is rapidly hydrolyzed under many ration conditions to ammonia. Under conditions of excessive intake, urea toxicosis is common.

Compounds resulting from the condensation of urea are primarily biuret, cyanuric acid and triuret. All of these are less soluble than urea and are less likely to cause ammonia toxicity under conditions where there is a shortage of readily fermentable carbohydrates. These compounds appear to offer many advantages over urea when low-quality forages are the primary source of dietary energy if, of course, the nitrogen supplied by these compounds can be utilized by the rumen microflora. This section considers some of the researches on these points.

Biuret: Berry (36) and Hatfield, et al. (37), have shown that biuret is less toxic to ruminants than urea and when high levels are included in diets, it is also more acceptable to ruminants than urea. Biuret is more stable than urea when used in ensiled mixtures.

The safety of biuret for ruminant feeding is due to its relatively slow rate of hydrolysis to ammonia (38,39,41). Biuret is hydrolyzed by biuretase (38,39,40,41), and possibly with the aid of urease, to ammonia and CO_2. The addition of readily fermentable carbohydrate, as with urea, will aid in protein synthesis.

Biuretase is an induced and intracellular enzyme (38,39,40,41) which requires an adaption period to reach its maximum rate of activity. In reviewing the research work which indicates a need for an adaptation period, one is struck by the variations in lengths of these periods, sometimes short and sometimes up to 70 days or more The results of Schroder and Gilchrist (40) offer an explanation: Using fistulated sheep to supply rumen fluid, they measured the ability of the fluid obtained from sheep exposed to different treatment to hydrolyze biuret. Rumen fluid seldom had any activity unless biuret had been previously included in the diets of the sheep for a period of time and up to the time the sample was taken. Sheep consuming hay which contained only 3.4 to 4.6% crude protein required 15 days to adapt to biuret (their rumen fluid gave peak biuretase activity 15 days after biuret was first added to their diets), while those receiving a medium nitrogen hay (5.9% C.P.) required 30 days and those receiving a 10.3% C.P. hay required 70 days.

When biuret is withdrawn from the diet, in a "de-adaptation" test (40), there was an abrupt decrease in biuretolytic activity, irrespective of diet. This discovery has been confirmed by many others and suggests that after de-adaptation, the animal must start over again for "re-adaptation" to dietary biuret. It is not known just how important re-adaptation is in the practical feeding of biuret or in feeding mixtures of biuret and urea (42). However, Thomas and Armitage (43) has reported that the feeding of a crude biruet product (42) to cattle every other day in comparison to every day feeding did not reduce cattle performance. This indicates that complete de-adaptation did not occur in those animals fed every other day. Many workers (43,44,45,46) have shown that biuret or mixtures of biuret and urea (42) can be used as a NPN source for beef and dairy cattle under a wide range of production requirements. The results of these researches provide beef and dairy cattle producers other alternatives to consider in choosing among the various feeding programs available to them. As biuret and mixtures of biuret and urea are more expensive per unit of nitrogen than urea, economics of the entire feeding operation along with certain risk factors must be considered in making a decision regarding which of the nitrogen sources to choose.

Cyanuric Acid and Triuret: Both of these compounds are produced in the controlled pyrolysis of urea to produce feedgrade biuret and can be utilized by ruminants with about the same efficiency as biuret. Economics do not dictate their production in a relatively pure state for feeding purposes.

Use of Ammonium Salts and Ammoniated Feeds

Many of the ammonium salts, which can be produced by various processes, have been fed and tested in cattle rations. Also, many different carbonaceous feeds have been ammoniated and fed to cattle. The purpose of this section is to report on some of the researches on these products:

Ammonium Salts: In an excellent review of the world literature on ammonium salts, Loosli and McDonald (47) concluded that these are well utilized, as would be expected, by cattle. However, urea is still the most economical source of NPN, thus the decision to use or reject ammonium salts is one of economics and availability.

Ammoniated Products: There are three major groups of compounds which have been ammoniated and tested and these are discussed as follows:

Molasses is high in readily fermentable carbohydrates and is low in nitrogen, therefore, it is an attractive compound for ammoniation. The results of Hershberger, et al. (48), have shown that only the "free" ammonia from these products is utilized by rumen microflora while that which is "bound" is not utilized. Also, the work of Tillman, et al. (49,50,51), and Davis, et al. (52), using growth and metabolism studies, demonstrated that the nitrogen from ammoniated molasses was not well utilized by cattle or sheep. Also, the author's group found that cattle and sheep fed ammoniated molasses become excited and will injure themselves. There were both peri- and endocardial hemorrhages in affected sheep. Other workers have reported that these lesions might have been produced by 4-Methlimidazale, which is produced in the ammoniating process. The possibilities of producing other toxic compounds are also good, thus it is doubtful if these products should be considered in the feeding of cattle.

Ammoniated Rice Hulls: Rice hulls will accept ammonia which can be utilized by cattle. When low levels of ammoniated rice hulls were compared to regular rice hulls to which urea was added at a level to make these isonitrogenous to the ammoniated product (53), utilization of these products by cattle did not differ but the urea-containing diet was more economical. When either 20 or 40% of the ammoniated product were fed to cattle receiving high-grain diets, digestive disturbances and decreased rates of gain were noted (54). Thus, this product should not exceed a level of 10% in high-grain rations and their use will be dictated by economical considerations.

Ammoniated Beet Pulp and Citrus Pulp: Several workers have found that the availability of nitrogen from ammoniated beet pulp is low. The author could find no experiments in which ammoniated beet pulp was compared to regular beet pulp, to which urea was added to make it isonitrogenous with the ammoniated product. Thus the economics of this product remain obscure.

Florida workers (55,56) reported that low levels of ammoniated citrus pulp can be fed to cattle with good results if grain is added to the total ration. However, high levels reduced gains of cattle. The reported experiments do not allow one to evaluate the availability of the added nitrogen in this feedstuff. Also, there have been no comparisons of the ammoniated product to a regular product containing urea; thus its economics also remain obscure.

Summary

Protein is the first-limiting nutrient in cattle production worldwide. Urea and other non-protein nitrogen (NPN) compounds, which can be hydrolyzed to ammonia, can be used to replace natural protein in cattle diets. Ammonia is the common denominator when the mode of action is determined for NPN compounds. A stepwise presentation of the hydrolysis and use of urea to produce microbial protein is used as an example of ammonia utilization. Urea is a widely used NPN compound; thus, factors which affect urea utilization were discussed as follows: 1) Level and solubility of dietary protein; 2) Kind of dietary carbohydrate; 3) Level of dietary fat; 4) Adequacy of dietary minerals and vitamins and presence of antibiotics and diethylstilbestrol; 5) The effect of rate and frequency of feeding.

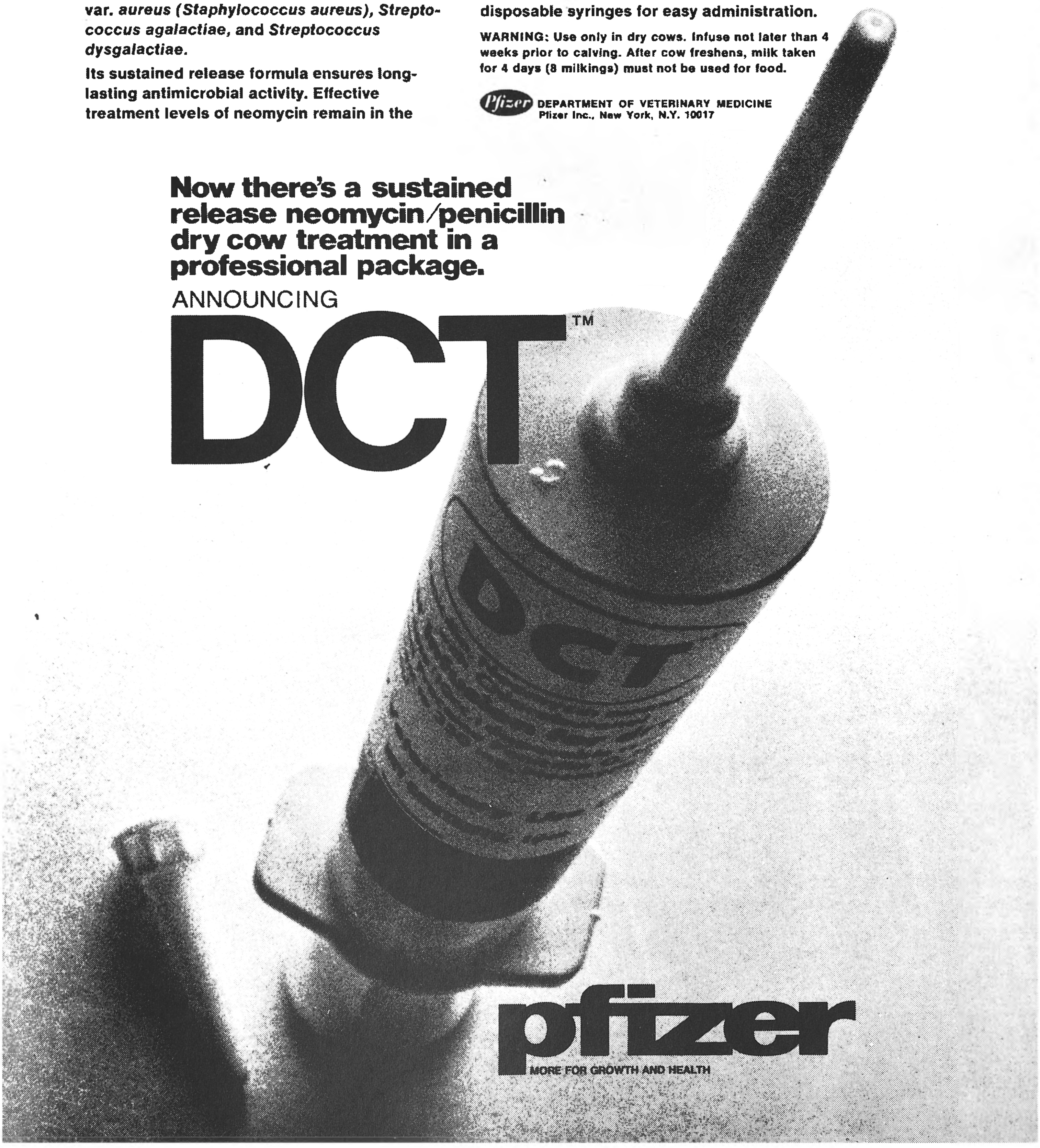
The practical usage of urea in beef and dairy cattle nutrition is discussed, with emphasis on ration conditions which promote urea utilization.

It was pointed out that urea, when fed at too high a level, will cause ammonia toxicity in cattle. Urea toxicosis is rapid and acute, but present experimental evidence does not support the idea that urea causes chromic toxicity in cattle. The clinical course of urea toxicity is described and suggestions for possible treatments were made. Oral administration of five to ten gallons of cold water appears to be one of the best treatments and reasons for its beneficial effects were discussed. There does not appear to be any effect of "near toxic" levels upon the incidences of abortions or upon the subsequent reproductive performance of cows which survive urea over-dosage.

The common misconceptions of veterinarians and livestock producers were considered and discussed. Also, the uses of biuret, ammonium sales and ammoniated products were discussed.

References

1. Weiske, H., H. Schrodt and S. V. Dangel. 1879. Z. Biol. 15:261. – 2. Hungate, R. E. 1966. The Rumen and Its Microbes. Academic Press, New York. 533 p. – 3. Bloomfield, R. A., G. B. Garner, and M. E. Muhrer, 1960. J. Anim. Sci. 19-1248 – 4. Lewis, D., K. J. Hill and E. F. Annison, 1957. Biochem. J. 66:587 – 5. Nolan, J. V. and R. A. Leng, 1972. Brit. J. Nutr. 27-177. – 6. Bryant, M. P. 1963. U.S.D.A. Agric. Res. Serv. Special Report 44-92, Washington, D.C. – 7. Hoogenrood, N. J. and J. R. Hird, 1970. Brit. J. Nutr. 24-119. – 8. Smith, R. H. and A. B' McAllen, 1970. Brit. J. Nutr. 24:545. – 9. Oltjen, R. R. and P. A. Putnam, 1966. J. Nutr. 89:385. – 10. Driedger, A. and E. E. Hatfield, 1972. J. Anim. Sci. 34:465. – 11. Nimrick, K., A. P. Peter and E. E. Hatfield, 1972. J. Anim. Sci. 34-488. – 12. U. S. Dept. Agr. Economic Res. Service, 1970 Feed Statistic Supplement to Statistical Bull. 446. – 13. Reid, J. T. 1953. J. Dairy Sci. 36-955. – 14. Burroughs, W., G. S. Ternus, A. H. Trenkle and R. L. Vetter, 1971a. Iowa State Univ. Exp. Sta. A. S. Leaflet R 152. – 15. Burroughs, W., A. Trenkle and R. L. Vetter, 1971b. Iowa State Univ. Exp. Sta. A. S. Leaflet R 151. – 16. Burroughs, W., A. H. Trenkle and R. L. Vetter, 1972. Iowa State Univ. Ext. Serv. EC 7771. – 17. Klett, R. H. 1972. Proc. AFMA Liquid Symposium, American Feed Manufacturers' Association, Arlington, Virginia. – 18. Karr, M. R., U. S. Garrigus, E. E. Hatfield and H. W. Norton, 1965. J. Anim. Sci. 24:459. – 19. Karr, M. R., U. S. Garrigus, E. E. Hatfield, H. W. Norton and B. B. Doane, 1965. J. Anim. Sci. 24:469. – 20. Williams, D. L., J. V. Whiteman and A. D. Tillman, 1969. J. Anim. Sci. 28:807. – 21. Pigden, W. J. 1971. Converence of FAO Ad Hoc. Com. on Anim. Nutr., Kampala, Uganda. FAO, Rome. – 22. Helmer, L. Y. and E. E. Bartley, 1971. J. Dairy Sci. 54:25. – 23. Tillman, A. D., G. M. Sheriba and R. J. Sirny, 1965. J. Anim. Sci. 24:1140. – 24. Smith, G. S. and E. E. Hatfield, 1967. Influence of Urea on Vitamin A Nutrition. In: Urea as a Protein Supplement M. H. Briggs, ed., Perganon Press, New York, 466 p. – 25. Rys, R. 1967. Urea in rations for dairy cows. In: Urea as a Protein Supplement M. H. Briggs, ed., Pergamon Press, New York, 466 p. – 26. Archibald, J. G. 1943. Mass. Agr. Exp. Sta. Bull. 406. – 27. Colenbrander, V. F., L. D. Muller, T. G. Martin and W. W. Carlton, 1968. J. Dairy Sci. 51:979. – 28. Patton, R. S., D. L. Hill, and R. E. Erb, 1970. J. Dairy Sci. 53:653. – 29. Virtanen, A. I. 1966. Science 153:1603. – 30. Oltjen, R. R. 1967. Proc. Cornell Nutr. Conf., Ithaca, New York. – 31. Lewis, D., K. J. Hill and E. F. Annison, 1957. Biochem. J. 66:587. – 32. Word, J. D., L. C. Martin, D. L. Williams, E. I. Williams, R. J. Panciera, T. E. Nelson and A. D. Tillman, 1969. J. Anim. Sci. 29:786. – 33. Buck, William B. 1972. Proc. Second Annual AFMA Liquid Feed Symposium. Am. Feed Manufacturers' Association, Arlington, Virginia. – 34. Floyd, W. E. 1970. Chemical and Metabolic Aspects of Urea-Ammonia Toxicoses in Cattle and Sheep. Ph.D. Thesis, Iowa State Univ., Ames, Iowa. – 35. Ryley, J. W. and R. J. W. Gartner, 1968. Proc. Australian Soc. Anim. Prod. 7:385. – 36. Berry, W. T., J. K. Riggs, and H. O. Kunkel, 1956. J. Anim. Sci. 22:139. – 37. Hatfield, E. E., U. S. Garrigus, R. M. Forbes, A. L. Newmann and William Gaiter, 1959. J. Anim. Sci. 18:1208. – 38. Bauriedel, W. R. 1971. J. Anim. Sci. 32:704. – 39. Bauriedel, W. R., L. F. Craig, J. C. Ramsey and E. O. Camehl, 1971. J. Anim. Sci. 32:711. – 40. Shroeder, H. H. E. and R. M. C. Gilchrist, 1969. J. Agric. Sci. (Cambridge) 72:1. – 41. Tiwari, A. D. 1972. Metabolism of Biuret by Ruminants. Ph.D. Thesis, Univ. of Ill., Urbana, Ill. – 42. Kedlor 230. Trademark of a feed grade biuret. The Dow Chemical Co., Midland, Mich. – 43. Thomas, O. O. and J. Armitage, 1972. J. Anim. Sci. 34:905. – 44. Nichols, N. E., A. L. Tesperance and W. E. Behrens, 1972. J. Anim. Sci. 34:896. – 45. Ammerman, C. B., G. J. Verde, J. E. Moore, W. C. Burns and C. F. Chicco, 1972. J. Anim. Sci. 23:121. – 46. Raleigh, R. J. 1972. Prac. Nutr. 6(4):16. – 47. Loosli, J. K. and I. W. McDonald, 1968. FAO Agric. Studies Bull. 75. Food and Agriculture Organization, Rome. – 48. Hershberger, T. V., O. G. Bentley and A. L. Moxon, 1959, J. Anim. Sci. 18:663. – 49. Tillman, A. D. and R. W. Swift, 1953. J. Anim. Sci. 12:201. – 50. Tillman, A. D., W. D. Gallup, L. S. Pope, G. H. McLaren and W. Price, 1957. J. Anim. Sci. 16:179. – 51. Tillman, A. D., W. D. Gallup and W. Woods, 1957. J. Anim. Sci. 16:419. – 52. Davis, R. F., R. H. Wasserman, J. K. Loosli and G. H. Grippin, 1955. J. Dairy Sci. 38:677. – 53. Tillman, A. D., R. D. Furr, K. R. Hansen, L. B. Sherrod and J. D. Word, Jr. 1969. J. Anim. Sci. 29:792. – 54. White, T. W. 1966. J. Anim. Sci. 25:25. – 55. Kirk, W. G., F. M. Peacock, E. M. Hodger and W. A. Jones, 1957. Fla. Agr. Exp. Sta. Annual Rpt., p. 333. – 56. Davis, G. K., K. Boney, R. B. Becker and P. T. D. Arnold, 1946. Fla. Agr. Exp. Sta. Annual Rpt. p. 58.


Now there's a sustained release neomycin/penicillin dry cow treatment in a professional package.
ANNOUNCING
DCT™
DCT
pfizer
MORE FOR GROWTH AND HEALTH