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Urine pH and DCAD: what, when and how?

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In a dairy cows' lactation cycle, the transition from non-lactating pregnant status to non-pregnant lactating status, represents the most critical phase which, if not accurately managed, might results in one or more periparturient diseases. Although our understanding of the transition period and its relationship with periparturient diseases has greatly increased, thus reducing tremendously the incidence of clinical cases, subclinical diseases still pose a threat for dairy farms profitability. Periparturient disease-associated culling and reproductive infertility, together with the obvious health and production concerns, are driving interest in predicting potential risks for disease.

Hence, scientists and animal technicians have been focusing on diseases prevention by managing the transition period and further developing technical methods to assess the cow's metabolic status. Metabolic profiling, although very accurate, is still an expensive method to assess the effectiveness of the transition programs applied; nevertheless, few cost-effective on farm tests have been available to predict the risk level of certain postpartum diseases. For instance, urine pH has been commonly used to assess the risk of hypocalcemia (also known as milk fever) postpartum. How does urine pH estimate the risk of milk fever? To answer this question we have to step a little bit backward and understand milk fever and the role pH in preventing it. Calcium (Ca) metabolism is regulated by two hormones: calcitonin and parathyroid hormone. During the dry period the only Ca lost is due to fetal absorption (4 to 6 g/day) and excretion (5 to 7 g/day), therefore the mechanisms for restoring plasma Ca levels are relatively inactive with calcitonin stimulating Ca storage in the bones and increasing excretion of the excess Ca. Soon after parturition the demand for Ca dramatically increases, because of colostrum (about 2 g Ca/L) and milk secretion (about 1.2 g Ca/L), causing a status of temporary hypocalcemia which triggers the parathyroid hormone (PTH) to activate the mechanisms of mobilization of Ca from the bones and its reabsorption from the renal tubules. Furthermore, there seems to be a refractory period of 48-72 hours during which, despite adequate levels of PTH, Ca mobilization doesn't occur due to the earlier calcitonin opposite mechanism. During this refractory period, especially high yielding cows can be faced with clinical or subclinical hypocalcemia with serious economic impact on the dairy enterprise. To overcome this problem, different nutritional strategies have been developed, with the modification of Dietary Cation Anion Difference (DCAD) being the most used strategy for the past 20 years. DCAD equations have been developed to estimate the relative balance between the principle cations and anions in the cow's diet. This equation is based on an initial observation from Scandinavian researchers which noticed that diets low in ash (with potassium known to be its main component) reduce the incidence of milk fever.

Further studies proved that diets low in potassium (and sodium) were acidogenic and stimulated Ca uptake from the bone reserves. In fact, when the pH of the intracellular fluid compartment is high (relative metabolic alkalosis), bone and possibly kidneys are refractory to the effects of PTH. Likewise, the stimulatory effects of PTH are enhanced during metabolic acidosis. In other words, diets high in strong cations during the transitional phase, especially sodium and potassium, tend to induce a metabolic alkalosis and milk fever can follow; whereas diets high in strong anions, primary chloride (Cl) and sulphur (S), will induce a relative metabolic acidosis and will prevent milk fever. Hence, the difference between the number of cations and anions absorbed from the diet determines the pH of the blood.

According to a recent review from Erdman R. and Iwaniuk M. (2017), of the various DCAD equations that have been developed, the one by Ender et al. (1971), although it probably overestimates the role of sulphur, is still the most commonly used by dairy nutritionists. The authors however indicated the NRC (2001) equation as probably more precise since it takes into account the relative absorption rate of each of the minerals in the equation; and the one developed by Goff et al. (2004) as the most effective taking into account the effectiveness of sulphur salts in reducing urine pH. When formulating a diet in NDS, the software will calculate the DCAD following the equations from Ender et al. (1971), Horst et al. (1997) and Goff et al (2004).

[Na + K] - [Cl + S]	+25,4
[Na + K + 0.15Ca + 0.15Mg] - [Cl + 0.6S + 0.5P]	+22,7
[Na + K] - [Cl + 0.6S]	+31,2

Because of the strong negative correlation between urine pH and net acid excretion (r^2=0.95) by cows fed anionic salts, the urinary pH evaluation becomes a useful tool to assess the degree of metabolic acidosis imposed by the DCAD (Rerat et al.,2014; Vagnoni et al., 1998; Goff et al.,1997). On this basis, NDS (E. Charbonneau et al.) is able to estimate the urine pH according to the DCAD of the diets. Thus, although anions concentration in feeds is not often measured, to obtain an effective DCAD concentration when formulating a diet, it is important to verify that the concentration of sodium and potassium (along with Cl and S) reported for the feeds of the NDS feed library reflects their actual content in the feeds available at the farm.

So, having accurate feeds characterization and having a diet formulating software that can calculate the DCAD and estimate the urine pH for us, what do we actually need to know?. Our responsibility is to know the **"what, when and how".**

What (DCAD to feed): Most forage-based diets have a DCAD of +15 to +35 mEq/100g with a normal urine pH of a dairy cow, like for any other herbivore, generally above 8, meaning that the animals are normally on a mild metabolic alkalosis. Due to the 72hours needed by the body to start mobilizing Ca, research has suggested to induce Ca mobilization slightly before parturition. Past research has suggested a pre-partum DCAD of -15 to -20 mEq/100g with a urine pH of 5.8 to 6.2 to induce and maintain a compensable metabolic acidosis to prevent milk fever (Roche et al., 2007); however, according to other researchers (Goff et al., 2019; Melendez and Poock 2017; Goff 2014; Charbonneau et al., 2006) the suggested DCAD pre-partum should be between 0 and -10mEq/100g and reach a urine pH between 6.2 and 6.8. The proposed DCAD is the results of several studies proving that lowering the DCAD from +25 to 0 mEq/100g (and the urine pH from 8 to 7) has a tremendous impact in reducing the modelled incidence of clinical milk fever from 16.4 to 3.2%; whereas lowering the DCAD to -15 mEq/100g would only reduce the modelled incidence of milk fever to 2.8% while providing excessive acidification and lower palatability of the diet. According to a recent study from Wang and collaborators (2018) excessive acidification may lead to a more severe metabolic acidosis with liver and kidney damages.

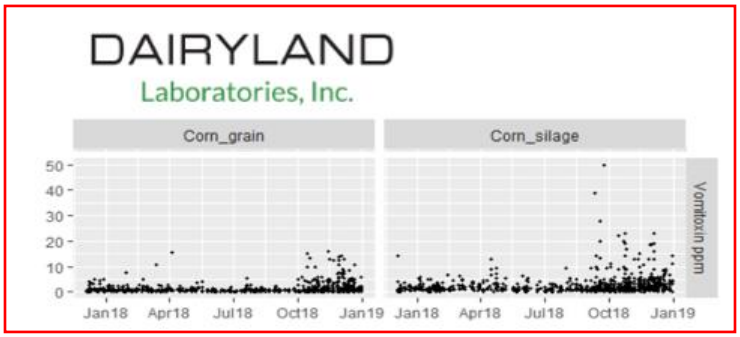
Although no minimal DCAD requirements have been established for lactating cows (mainly due to lack of research data), from a recent meta-analysis of DCAD effects during lactation (Iwaniuk and Erdman, 2015), it seems that a practical DCAD range would be between 0 and 50mEq/100g. It would appear, in fact, that maximum DMI is achieved with DCAD between 29 and 42,5 mEq/100g, whereas a 3.5%FCM response is maximized with DCAD concentration around 50 mEq/100g. These values can be explained by the fact that DCAD between 0 and 50 mEq/100g increase ruminal pH from 6.3 to 6.5 and increase DM digestibility by increasing NDF digestibility of 7.5 percentage unit (Erdman and Iwaniuk, 2017). On the base of the abovementioned information, when manipulating the DCAD concentration of the lactation diet, besides considering the possible advantages of using the higher DCAD concentration, the farm specific case should be considered to formulate a cost-effective diet.

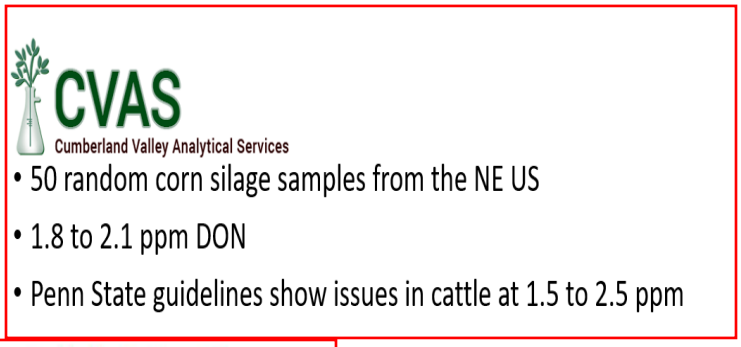
When (to feed negative DCAD): We've mentioned already that an excessive negative DCAD may lead to more severe metabolic acidosis with health and economic implications. However, it is not only the concentration of DCAD to play a role but also for how long a diet with negative DCAD is fed. In fact, we did mention that, to overcome the issue of the 72 hours refractory period, the negative DCAD needs to be fed per-partum; furthermore, recent studies have proved that it is necessary to feed an acidogenic diet for at least 9 days before observing effective changes in the urine pH. However, feeding a negative DCAD for more than 40 days negatively affects the performance of the cow and its offspring. In fact, both the dam and the offspring can develop uncompensated metabolic acidosis; in addition, the metabolic acidosis will not only alter the blood pH but will alter the composition of the follicular fluid affecting the oocytes viability and, thus, disrupting the animal's fertility.

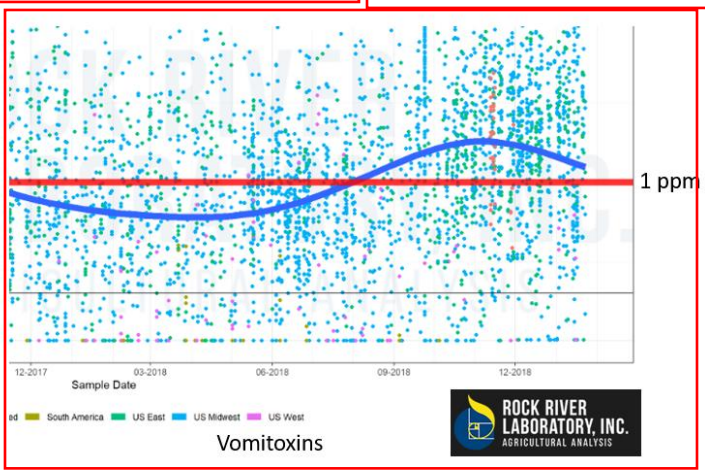
How (to check if DCAD works; to choose the right DCAD formula): Although an indication of urine pH that needs to be achieved during the close-up period has been given, it really depends more on how much we lower the urine pH rather than to which value. In fact, reducing the urine pH from 8 to 7 might already provide the expected results (Charbonneau et al., 2006). Therefore, knowing the urine pH of the dry cows (a good estimation of the all group is obtained by testing the midstream urine, using pH test strips, of about 20% of the cows in the group) and testing it again at least 9 days after the introduction of the acidogenic diet could be an ideal method to assess the effectiveness of the new diet.

When formulating a diet with NDS, which DCAD value should we take into account? Although different equations would estimate different DCAD concentrations, research has showed that to prevent milk fever, the DCAD concentration range during close-up is relatively ample (0 to -10 mEq/100g). Recently, an even more ample range has been suggested for lactating cows (0 to 50 mEq/100g), however, more research is needed to better understand how precisely DCAD concentrations need to be modulated to maximize its effectiveness.

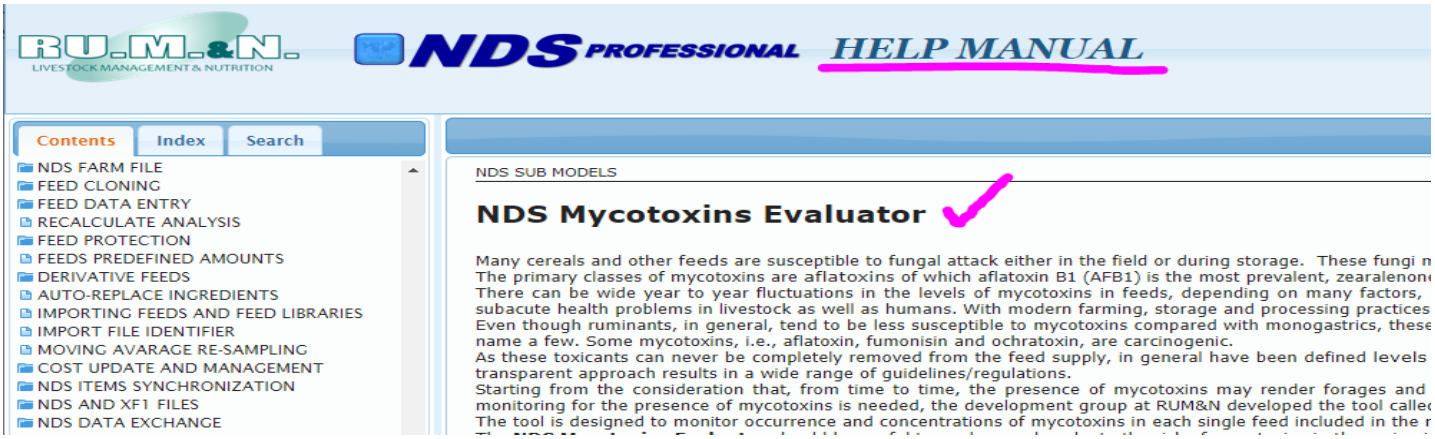
Messages and discussions with many consultants it seems that many parts of the NE had challenges getting corn silage put up in a timely manner. Many fields were extremely wet, and the plant went past dry down points and much of the harvest was on extremely dry plants that might have had more external moisture on it. The discussion has been about inputting these dry forages and the load of Mycotoxins in these forages. Testing has shown higher levels this fall







Great opportunity to use the Mycotoxin Evaluator in NDS to check possible issues with all Mycotoxins. From the Manual you can access the NDS Mycotoxin Evaluator tutorial and learn how this tool can show the loads of mycotoxins in rations to cattle.



Send us your comments on this topic! Dave Weber is at rumendvm@gmail.com; RUM&N Staff is at info@rumen.it

Note that the features and utilities developed by the NDS team described above are not components of the underlying CNCPS model, and do not change the CNCPS outputs or results. *Questions about use of these features should be directed to the NDS support team, and not to the CNCPS group at Cornell.*

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