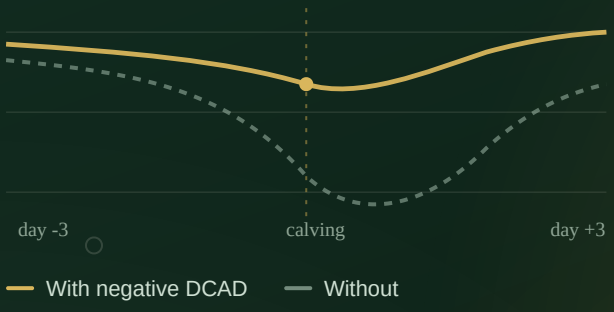
TECHNICAL REVIEW

Anionic Feeding.

How **negative DCAD** in the close-up ration prevents hypocalcemia and protects transition cow health — an evidence-based field guide.

BLOOD CA²⁺ AROUND CALVING



Prepared by Tolga Akay · September 2026

Based on peer-reviewed journal articles & university extension research

DCAD · HYPOCALCEMIA · TRANSITION HEALTH

CONTENTS

In This Report

01 Introduction — Why Is the Close-up Period So Critical?

02 What Is DCAD? Formula, Units, and Target Values

03 How Anionic Feeding Works: The Mechanism

04 Urine pH: When, How, and Why to Measure It

05 Continuing Anionic Feeding Postpartum: Is the "4-5 Day" Rule Correct?

06 What to Watch For in the Close-up Ration

07 Preventing Metabolic Disease: A Chain of Consequences

08 "Activating" Calcium Metabolism

09 Holstein vs. Jersey: Why the Approach Should Differ

10 Practical Summary and Checklist

— References

How this review was prepared

This report was compiled by reviewing meta-analyses, controlled trials, and review articles from the Journal of Dairy Science, Animal (Cambridge), MDPI journals, the PubMed/PMC database, and dairy extension units at universities such as Cornell, Washington State, Illinois, and Utah State. Points that are not numerically settled and are reported in the literature only as trends are explicitly flagged in the text as "schematic" or "general trend." All sources are listed in the references section at the end.

01 — INTRODUCTION

Why Is the Close-up Period So Critical?

The **close-up period**, beginning roughly three weeks before calving, is the final and most delicate stage of the dry period. The quality of feeding during this three-week window determines, far more than calving itself, how well the cow will tolerate the physiological "shock" she faces in the first 24-72 hours after calving.

At calving, the cow suddenly begins transferring large amounts of calcium to the mammary gland for colostrum and, immediately after, for milk production. Daily calcium requirement/loss during the dry period is around 10 g; with calving, this figure roughly triples to about 30 g/day within 24 hours. The mechanisms that maintain calcium homeostasis (the parathyroid hormone axis, bone resorption, intestinal absorption) cannot adapt to this sudden demand within seconds — they need days. It is precisely this adaptation lag that close-up feeding is designed to address.

The **hypocalcemia** (low blood calcium) that develops in an inadequately prepared cow is not limited to the clinical picture known as "milk fever"; as detailed in later sections, it is also a trigger for a range of metabolic and reproductive problems, including retained placenta, metritis, ketosis, fatty liver, and displaced abomasum. For this reason, many field veterinarians and nutritionists describe the close-up period as "the period that determines the fate of the lactation."

> **The focus of this report**
>
> This report examines, based on current literature and with directly applicable field recommendations, how **anionic (negative DCAD) feeding** programs applied during the close-up period work, how they are monitored (particularly via urine pH), their relationship to metabolic disease, and how they differ across breeds.

02 — CORE CONCEPT

What Is DCAD? Formula, Units, and Target Values

DCAD (Dietary Cation-Anion Difference) is the difference, expressed in milliequivalents (meq), between the strong cations in the diet (sodium and potassium) and the strong anions (chlorine and sulfur):

$$\text{DCAD (meq)} = (\text{Na}^{+} + \text{K}^{+}) - (\text{Cl}^{-} + \text{S}^{2-})$$

Rations fed during most of the dry period (the far-off period) are naturally positive in DCAD, since forages are generally rich in potassium. During the close-up period, the goal is to deliberately shift this balance **negative** by adding chloride- and sulfate-based **anionic salts** to the ration (such as calcium chloride, magnesium chloride, ammonium chloride/sulfate, or commercial HCl-based products).

Program type	Approximate target DCAD	When it is preferred
Full DCAD	−10 to −15 meq/100 g DM *(≈ −100 to −150 meq/kg DM)*	Herds with a history of milk fever, high-risk multiparous cows, operations able to monitor closely
Partial DCAD	Around 0 to −5 meq/100 g DM	Herds with low/controlled forage potassium wanting a more measured approach; usually paired with a supplemental calcium strategy

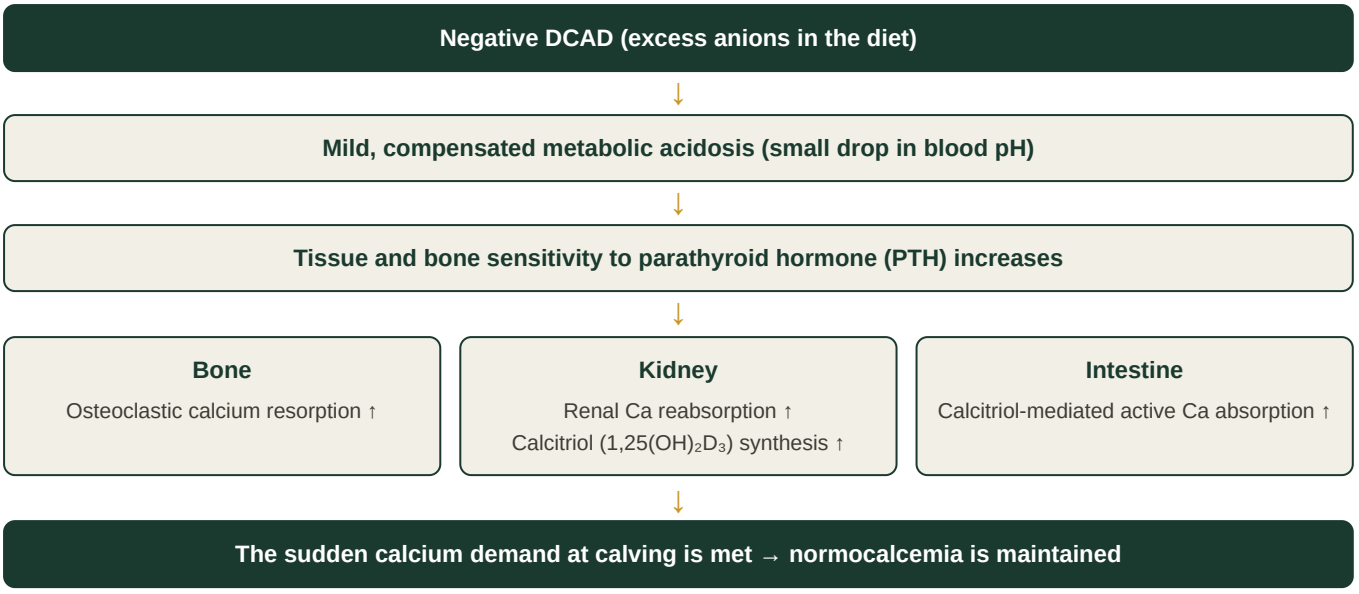
Note: some sources report DCAD in meq/kg DM, others in meq/100 g DM — always check the unit when reading a report; misinterpretations off by a factor of ten stem from this confusion.

03 — MECHANISM

How Anionic Feeding Works

Understanding how anionic feeding "activates" calcium metabolism matters both for interpreting urine pH monitoring correctly and for understanding why the program sometimes fails to work (e.g., in the presence of low magnesium).

When dietary DCAD is shifted negative, the cow enters a state of mild, **compensated metabolic acidosis** — meaning blood pH shifts slightly toward the acidic side without a clinically significant drop. This small but effective change triggers a cascade of events in the calcium homeostasis system:



In a cow with a healthy, balanced DCAD, this "pre-priming" does not occur, because tissue is already adequately sensitive to PTH; the calcium mobilization system can then lag behind the sudden demand at calving. The literature sometimes refers to this state as resembling "diet-induced pseudohypoparathyroidism" — that is, even when PTH is sufficiently elevated in the blood, tissues fail to respond to it quickly enough.

> **Magnesium's overlooked role**
>
> Intracellular signal transduction at the PTH receptor is magnesium-dependent. If dietary magnesium is insufficient, PTH's effect remains weak regardless of how negative the DCAD is, and hypocalcemia may not be prevented. Ensuring an adequate, **highly bioavailable** magnesium source in close-up rations is one of the prerequisites for a DCAD program to work.

How does it lower urine pH? The excess chloride and sulfate ions from anionic salts create a mild acid load in the blood that the kidneys must excrete. The kidneys eliminate this excess acid (hydrogen ions) through the urine to keep blood pH close to normal — and this process is reflected as a measurable drop in urine pH. In other words, **urine pH is not a "mirror" of the blood's acid-base status; it is an indicator of the kidney's response to balancing that status.** This distinction underlies the monitoring logic discussed in the next section.

Urine pH: When, How, and Why to Measure It

A critical distinction: urine pH is not the same thing as milk fever risk

The large-scale meta-analysis by Lean and colleagues (2006) found a **linear** relationship between DCAD and milk fever risk (the more negative the DCAD, the lower the risk), while showing that the relationship between DCAD and urine pH is **not linear but curvilinear**. In the authors' own words, urine pH *"monitors the efficacy of the DCAD; it does not monitor milk fever risk directly."* Urine pH should therefore be used not as a "diagnosis," but as a **process-control tool** that indicates whether the ration is being correctly formulated and consumed.

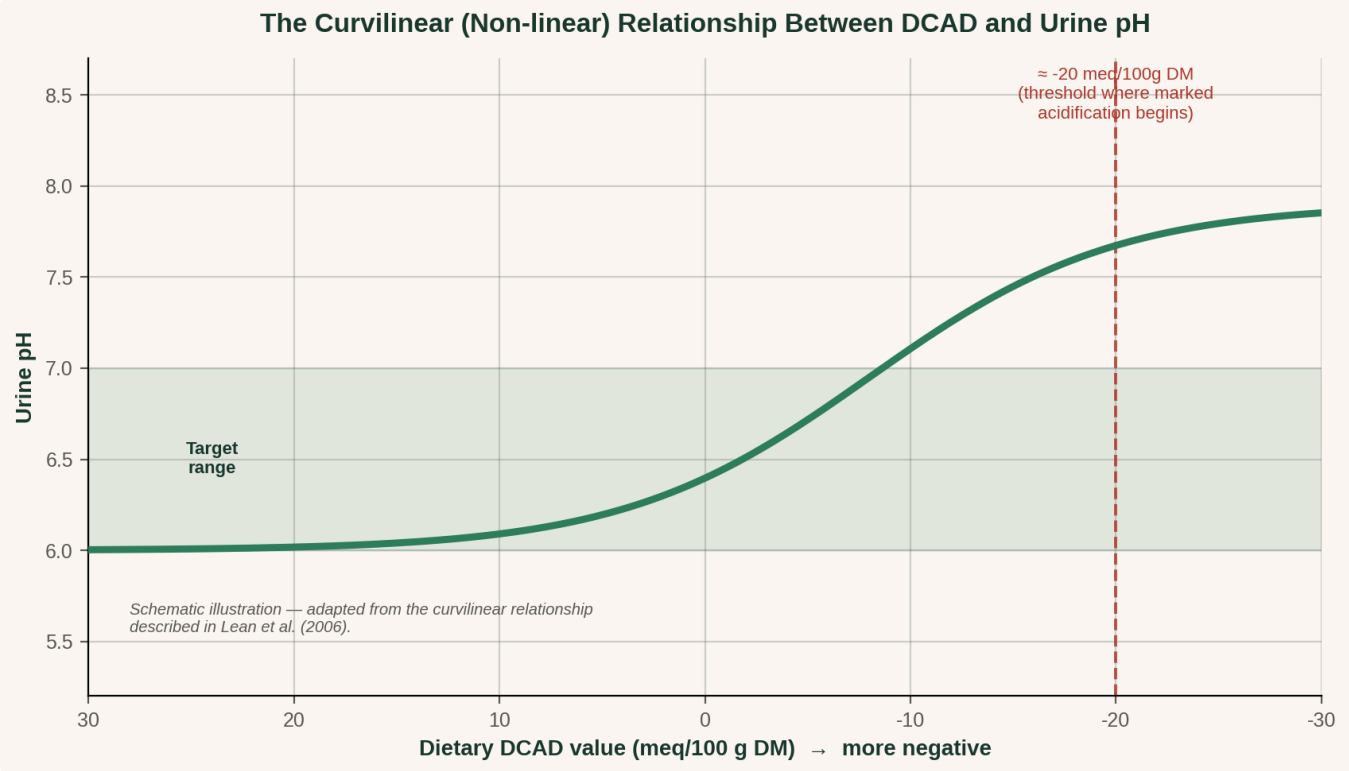


Figure 1 — As DCAD becomes more negative, urine pH first declines slowly, then drops more sharply from around −20 meq/100 g DM onward (schematic illustration adapted from Lean et al., 2006).

How many days later should it be measured?

Urine pH should not be measured immediately after a ration change, but rather **at least 3 days after the animal switches to the close-up ration**. This is because the kidney needs a few days to adapt to the new acid-base load and for urine pH to reach a "steady state." Measurements taken earlier can give a misleading picture of the ration's true efficacy.

Practical measurement protocol

- ✓ Begin measuring **at least 3 days** after animals switch to the close-up ration.
- ✓ Select **10 cows at random** from the group (excluding animals very close to calving or that are sick).
- ✓ Discard the highest and lowest readings and **average the remaining 8 values**; do not base decisions on a single animal's result.
- ✓ Test at a consistent time of day where possible; measure the fresh urine sample **on the farm, immediately** — samples sent to or held for a laboratory can show falsely elevated pH due to bacterial ammonia production.
- ✓ Weekly checks for large herds and monthly checks for smaller operations are a practical starting point.

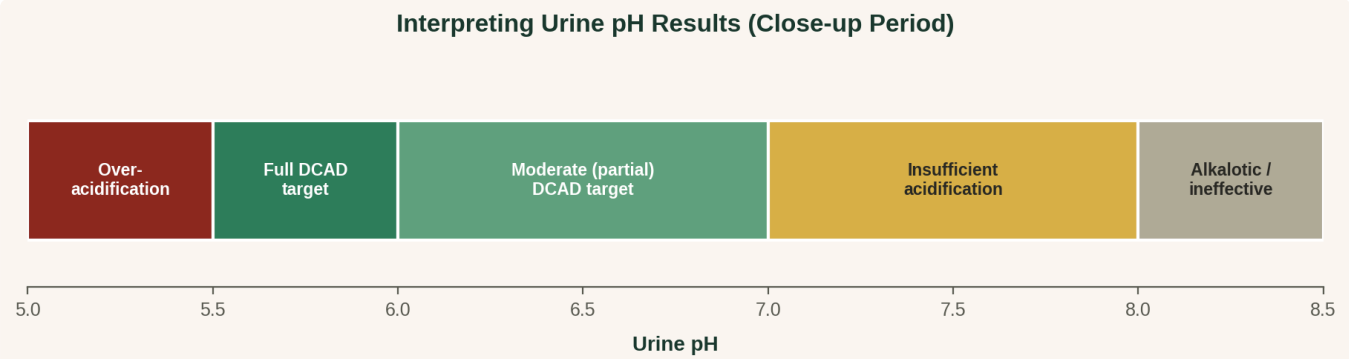


Figure 2 — General interpretation of urine pH results as a group average. The classic target for full DCAD programs is 6.0-6.5; a range of 6.0-7.0 is also considered acceptable for partial DCAD programs.

A newer finding: targets may vary by parity

An analysis published in 2025, covering 660 multiparous Holstein cows from 9 studies, found that the relationship between prepartum urine pH and postpartum blood calcium **differs by parity group**: in 2nd- and 3rd-lactation cows, lowering pH below 7.75 was sufficient, with no additional benefit from further acidification beyond that; in cows in their 4th lactation or older, the optimal range was narrower (approximately 6.26-6.75), and results worsened below 5.75 or above 7.25. The same study also found that the calcium "nadir" occurred within the first 24 hours after calving in 75% of cases. Practical takeaway: rather than a single "magic number," it may be more accurate to also account for the herd's parity distribution.

05 — FREQUENTLY ASKED QUESTION

Continuing Anionic Feeding Postpartum: Is the "4-5 Day" Rule Correct?

A recommendation commonly heard in the field is to continue anionic feeding for another 4-5 days after calving. There is no clean "yes" or "no" answer to this question; the literature paints a nuanced picture:

The classic approach

In standard practice, negative DCAD is discontinued at calving and the cow is switched to a fresh-cow ration, because after calving the priority is no longer "mobilizing" calcium but maximizing appetite and dry matter intake (DMI). Unnecessarily extending anionic feeding — given its palatability problems — carries a risk of suppressing DMI in the fresh cow.

What does the literature say?

A controlled study in multiparous cows examining the effects of **extending** negative DCAD feeding into the postpartum period found that this practice **did not negatively affect** postpartum performance (milk yield, DMI). This is a finding that supports the cautious approach of "a few extra days won't hurt." However, the "priming" of the calcium homeostasis system occurs mainly during the **prepartum** 2-3 week window (see Section 3); the evidence that extending a few days postpartum provides a meaningful protective benefit **on top of** prepartum preparation is not as strong as the evidence that it does no harm.

Practical takeaway

The recommendation to continue for 4-5 days postpartum is mostly offered as a **safety margin**, particularly on operations where late calvings and pen transitions cannot be cleanly separated in practice. It should be viewed not as a mandatory rule but as an **optional safety margin** to be weighed against the farm's barn/pen layout and fresh-cow DMI monitoring. If extended, fresh-cow appetite and DMI should be closely monitored.

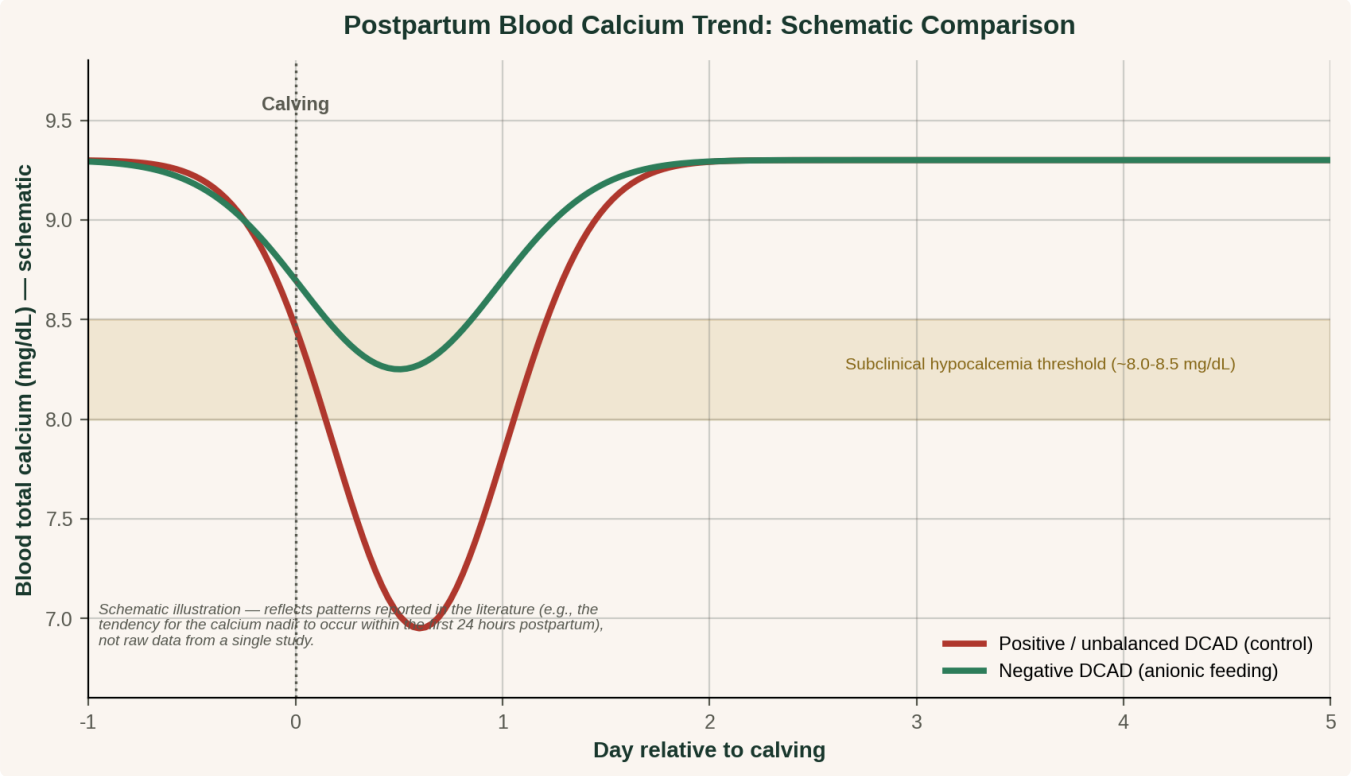


Figure 3 — The tendency for the calcium "nadir" to occur around calving (particularly within the first 24 hours); cows fed negative DCAD are expected to show a shallower, shorter-lived nadir (schematic illustration).

06 — APPLICATION

What to Watch For in the Close-up Ration

The success of an anionic feeding program depends not only on the additive itself but also on the mineral profile of the **base ration** (the base forages and concentrates). The points below are the most common causes of field failures.

Points requiring caution

- ✕ **High-potassium forages** (some pasture grasses, fresh pasture, mature alfalfa) — potassium pulls DCAD strongly toward positive, working against the anionic salts.
- ✕ **Sodium bicarbonate and similar buffers** — useful in lactating rations, but they raise DCAD in the close-up ration, working against the goal.
- ✕ **Uncontrolled high calcium** — some meta-analysis findings show that high calcium levels (around 1.1-1.5% of ration DM) can paradoxically increase milk fever risk when DCAD is unbalanced; brief exposure to high calcium is particularly risky.
- ✕ **High-bicarbonate/hard drinking water** — a frequently overlooked cation source in DCAD calculations; water analysis should not be neglected.
- ✕ **Untested forage from a single source** — potassium content can vary substantially between batches; calculating DCAD without regular feed analysis is unreliable.

Points worth supporting

- ✓ **Low-potassium base forages** (such as corn silage, wheat straw) should be preferred; use high-K forages in limited proportions when needed.
- ✓ **Adequate, highly bioavailable magnesium** — a prerequisite for PTH signal transduction (see Section 3).
- ✓ **An anionic salt source with tested palatability** should be chosen; sources (calcium chloride, magnesium chloride, ammonium salts, commercial HCl-based products) can differ in their effects on taste and DMI.
- ✓ **Regular feed and water analysis** to periodically confirm the ration's actual DCAD value; the formulated value can drift from the real value on the farm over time.
- ✓ **Adequate energy and DMI support** — since anionic salts can suppress appetite, the ration's palatability and energy density should be balanced accordingly.

Why does palatability matter?

Anionic salts are inherently bitter/unpalatable and can suppress dry matter intake when used in excess. Source selection and homogeneous distribution within the mixer (spread evenly through the whole ration without clumping) are therefore not just a formulation issue, but also a matter of **mixing and management**.

07 — CLINICAL CONNECTIONS

Preventing Metabolic Disease: A Chain of Consequences

The real power of anionic feeding lies not in preventing a single disease, but in cutting off, at its source, a **chain of diseases** that trigger one another. Each link in this chain is addressed separately below.

Retained placenta and metritis

Expelling the placenta is an active process requiring strong uterine muscle contraction, and this contraction is heavily calcium-dependent. In a hypocalcemic cow, myometrial (uterine muscle) contractile strength weakens, so the placenta is not expelled on time; this creates a favorable environment for bacterial contamination within the uterus, increasing metritis risk. Field data indicate that well-managed negative DCAD programs reduce the incidence of retained placenta and metritis.

Ketosis and fatty liver

The link between hypocalcemia and ketosis/fatty liver is indirect but strong: low blood calcium impairs smooth muscle function (including rumen motility), reducing dry matter intake. In a cow already in negative energy balance after calving, this additional loss of appetite accelerates fat mobilization from adipose tissue (rising NEFA); when the fatty acids reaching the liver exceed the liver's processing capacity, **fatty liver** develops, and as gluconeogenic capacity falls, **ketosis** develops as well. These two conditions feed a vicious cycle: fatty liver impairs gluconeogenesis, which deepens ketosis; ketosis further suppresses appetite, which increases fat mobilization. The literature reports that cows with subclinical ketosis have approximately **4.9 times** the risk of metritis and approximately **6.1 times** the risk of displaced abomasum compared with cows without it — which explains why hypocalcemia is described as a "gateway disease."

The link with mastitis: how strong is the evidence?

Calcium is required as an intracellular signaling molecule for neutrophil (the immune system's first line of defense) degranulation, phagocytosis, and the oxidative burst response; a theoretical weakening of immune function is therefore expected in hypocalcemic cows. To be candid, however, field studies have not always demonstrated a **consistent, statistically significant** relationship between negative DCAD programs and clinical mastitis incidence. In other words, while the mechanism is plausible, the claim that "anionic feeding directly reduces mastitis" is not as well supported by evidence as its effect on retained placenta/metritis — this is a point where overstated claims should be avoided.

Disease	Mechanism linking it to hypocalcemia	Effect of negative DCAD
Milk fever / subclinical hypocalcemia	Direct — insufficient PTH sensitivity and Ca mobilization	Strong, consistent reduction
Retained placenta / metritis	Weak uterine contraction → delayed placental expulsion	Findings point to a consistent reduction
Ketosis / fatty liver	Low DMI → increased fat mobilization	Indirect, positive contribution
Displaced abomasum	Reduced smooth muscle (rumen/abomasum) motility	Findings point to a reduction
Mastitis	Theoretical weakening of neutrophil function	Inconsistent / not well established

08 — PHYSIOLOGY

"Activating" Calcium Metabolism

Calcium homeostasis rests on a continuous balance between three organs: **bone** (the large calcium reservoir), the **kidney** (the center for excretion/reabsorption and calcitriol production), and the **small intestine** (the gateway for active absorption). In a cow under a healthy, balanced DCAD, these three systems operate "loosely" relative to the sudden demand at calving; the mild acidosis created by negative DCAD acts as a kind of **pre-warming**, engaging all three systems before calving. As a result, the cow enters the calcium crisis of calving not from a "standing start," but with a system that is already partially activated.

A further point worth emphasizing here is that the key variable is not the calcium supply itself but the **sensitivity of the system**. Indeed, the same meta-analysis data show that briefly offering high calcium in the ration (particularly when DCAD is unbalanced) can increase milk fever risk rather than reduce it — because an abundant calcium supply can "lull" the body's own mobilization mechanisms into inactivity. This is why anionic feeding should be viewed not merely as a mineral supplement, but as a **physiological priming strategy**.

09 — BREED DIFFERENCES

Holstein vs. Jersey: Why the Approach Should Differ

In the dairy literature, the Jersey breed is widely reported to be generally **more predisposed** to clinical hypocalcemia (milk fever) than Holsteins. Several factors may contribute to this:

- ✓ Jersey milk has a higher calcium content per unit volume than Holstein milk, meaning a relatively more intense calcium demand relative to body weight at the start of lactation.
- ✓ Baseline PTH sensitivity and the rate of bone calcium mobilization in Jersey cows have been reported to show breed-specific differences.
- ✓ Smaller body mass can mean that the same absolute calcium loss represents a relatively larger physiological burden.

As a practical consequence, many field specialists recommend being **less tolerant (erring on the safe side)** with DCAD targets in Jersey-heavy herds, increasing monitoring frequency, and paying closer attention to the calcium/magnesium balance during the close-up period. That said, this is a general trend; the final decision should always rest on the herd's own forage profile, water analysis, and history of hypocalcemia incidence — breed alone is not a sufficient decision criterion.

10 — SUMMARY

Practical Summary and Checklist

This section condenses the entire report into a checklist that can be applied quickly in the field.

- ✓ Start the close-up ration ~3 weeks before calving; target −10 to −15 meq/100 g DM for full DCAD, or around 0 to −5 meq/100 g DM for partial DCAD.
- ✓ Measure urine pH **at least 3 days after** switching to the ration, on a group basis (10 animals, extremes discarded); target 6.0-6.5 for full DCAD, 6.0-7.0 for partial DCAD.
- ✓ Use urine pH not as a "milk fever test," but as a check that the ration is correctly mixed and consumed.
- ✓ Keep high-potassium forages and uncontrolled calcium/sodium bicarbonate sources out of the close-up ration; do not neglect water analysis.
- ✓ Guarantee adequate magnesium — a prerequisite for DCAD to work at all.
- ✓ Extending anionic feeding for 4-5 days postpartum is not mandatory; if you choose to, apply it as an optional safety margin while closely monitoring fresh-cow DMI.

- ✓ Adopt stricter targets and more frequent monitoring in Jersey-heavy herds or herds with a history of milk fever.
- ✓ Do not overstate the effect on mastitis; the strongest and most consistent benefits of a DCAD program are on hypocalcemia, retained placenta/metritis, and, indirectly, ketosis/fatty liver.

> **Closing note**
>
> Anionic feeding is not a "recipe" on its own; it is a **system** made up of forage analysis, water quality, adequate magnesium, and regular urine pH monitoring. If any component of this system is neglected, a DCAD formulation that is correct on paper may still fail to deliver the expected result in the field.

REFERENCES

Sources Consulted

The sources below consist of peer-reviewed journal articles, meta-analyses, controlled trials, and university extension publications.

1. Lean, I.J., DeGaris, P.J., McNeil, D.M., Block, E. (2006). Hypocalcemia in Dairy Cows: Meta-analysis and Dietary Cation Anion Difference Theory Revisited. *Journal of Dairy Science*, 89(2):669-684. journalofdairyscience.org
2. Charbonneau, E., Pellerin, D., Oetzel, G.R. (2006). Impact of Lowering Dietary Cation-Anion Difference in Nonlactating Dairy Cows: A Meta-Analysis. *Journal of Dairy Science*. sciencedirect.com
3. Santos, J.E.P. et al. (2019). Meta-analysis of the effects of prepartum dietary cation-anion difference on performance and health of dairy cows. *Journal of Dairy Science*. sciencedirect.com / PubMed 30594362
4. Dietary Cation-Anion Difference Effects on Performance and Acid-Base Status of Lactating Dairy Cows: A Meta-Analysis. *Journal of Dairy Science*. sciencedirect.com / PubMed 15328236
5. Controlled trial of the effect of negative dietary cation-anion difference on postpartum health of dairy cows. *Journal of Dairy Science*. journalofdairyscience.org
6. Extended negative dietary cation-anion difference feeding does not negatively affect postpartum performance of multiparous dairy cows. *Journal of Dairy Science*. sciencedirect.com
7. Effect of a very low negative dietary cation-anion difference (DCAD) diet on plasma and urine metabolomics of prepartum Holstein cows. *JDS Communications / PMC*. pmc.ncbi.nlm.nih.gov
8. The association of prepartum urine pH, plasma total calcium concentration at calving and postpartum diseases in Holstein dairy cattle. *Animal*. sciencedirect.com
9. Associations between prepartum urine pH and periparturient blood calcium concentrations in multiparous Holstein cows. *Frontiers in Veterinary Science* (2025). frontiersin.org
10. Review: Dietary cation-anion difference to prevent hypocalcemia with emphasis on over-acidification in prepartum dairy cows. *Animal*. sciencedirect.com
11. Screening of Anionic Salts for Palatability, Effects on Acid-Base Status, and Urinary Calcium Excretion in Dairy Cows. *Journal of Dairy Science*. sciencedirect.com
12. Reducing prepartum urine pH by supplementing anionic feed ingredients: Effects on physiological and productive responses of Holstein × Gir cows. *Journal of Dairy Science*. sciencedirect.com / PubMed 30077445
13. The importance of DCAD and time in the close-up period on postpartum health and production of dairy cows. Washington State University Veterinary Medicine Extension (2024). vetextension.wsu.edu
14. Overton, T.R. Update on DCAD for Dry and Lactating Cows. Cornell University eCommons. ecommons.cornell.edu
15. The Most Important Metabolic Diseases in Dairy Cattle during the Transition Period. *Animals* (MDPI), 14(5):816 (2024). mdpi.com
16. Hypocalcemia in Dairy Cows: A Systematic Review of Metabolic Implications and Management Strategies. *Life* (MDPI), 16(7):1082. doi.org/10.3390/life16071082
17. Optimizing Calcium Metabolism in Transition Cows: A Pragmatic Review. *Biomedical Journal of Scientific & Technical Research*. biomedres.us
18. Effects of Postpartum Supplemental Oral Ca for Dairy Cows Fed Prepartum Dietary Acidogenic Salts. *PMC*. pmc.ncbi.nlm.nih.gov / PubMed 34827864
19. Effect of prepartum dietary cation-anion difference strategy and level of dietary calcium on postpartum blood calcium status and milk production of multiparous Holstein cows. *Journal of Dairy Science*. sciencedirect.com
20. Symposium review: Transition cow calcium homeostasis — Health effects of hypocalcemia and strategies for prevention. *Journal of Dairy Science*. sciencedirect.com
21. Dietary Cation Anion Difference — an overview. *ScienceDirect Topics*. sciencedirect.com
22. Measuring urine pH as an indicator of calcium balance. University of Illinois — Dairy Focus Newsletter. dairyfocus.illinois.edu
23. How to interpret urine pH results for pre-fresh cows. *Progressive Dairy*. agproud.com
24. 7 steps to start a moderate DCAD program. *Progressive Dairy*. agproud.com
25. Target ration DCAD levels, not urine pH results. *Progressive Dairy*. agproud.com
26. Dairy Nutrition: Dietary Cation-Anion Difference Update. *Engormix*. engormix.com
27. Importance of a Dietary Cation-Anion Difference in Peripartum Dairy Cows. Utah State University Extension. extension.usu.edu
28. Dietary Cation-Anion Difference for Dairy Rations. DAIReXNET / eXtension. dairy-cattle.extension.org
29. Jersey vs. Holstein: Why the Difference Matters for Nutrition and Performance. Jefo. jefo.ca
30. Nutrition of Jersey Cows — Little Holstein Cows or a Breed Apart? Ohio State University, Buckeye Dairy News. dairy.osu.edu
31. Prevention and treatment of milk fever. University of Minnesota Extension. extension.umn.edu

This report is intended for educational and professional information-sharing purposes; for a farm-specific ration formulation, consultation with a veterinarian or ruminant nutritionist is recommended.