



Transition Period Nutrition in Dairy Cows: Metabolic Adaptation and Disease Prevention

*Dr. Hrishikesh B Gudaghe

PhD Scholar, ICAR-Indian Veterinary Research Institute, Izatnagar,
Bareilly, Uttar Pradesh, India

*Corresponding Author's email: hrishi610@hotmail.com

The transition period in dairy cattle, conventionally defined as the three weeks preceding calving through the first three weeks of lactation, represents the single most metabolically demanding phase of the production cycle. During this narrow window, the cow must simultaneously complete fetal growth, initiate colostrogenesis and lactogenesis, and adapt her metabolism, endocrine profile, and immune function to support an exponential rise in nutrient output. It is estimated that a large majority of all clinical disease in the dairy herd, including displaced abomasum, clinical ketosis, milk fever, retained placenta, and metritis, occurs in the first month after calving, a pattern that reflects the failure of many cows to adapt successfully to the metabolic load of early lactation. For the practicing nutritionist and veterinarian, understanding the biochemical basis of transition failure, and applying evidence-based feeding strategies during the close-up and fresh periods, is therefore central to herd health, reproductive performance, and farm profitability.

Physiological and Metabolic Basis of the Transition Period

As parturition approaches, dry matter intake declines by as much as 30 percent in the final week of gestation, a phenomenon attributed to reduced abdominal space from the gravid uterus, elevated circulating estrogen, and inflammatory signaling associated with fetal cortisol release. This intake depression coincides with a sharp rise in nutrient demand once lactation begins, since a cow producing 40 kilograms of milk daily requires roughly two to three times the glucose, amino acid, and fatty acid supply needed during the dry period. The result is an obligatory period of negative energy balance (NEB) that essentially all dairy cows experience to some degree, with the depth and duration of NEB determining whether a cow transitions smoothly or develops metabolic disease. Cows mobilize adipose tissue to meet the energy deficit, releasing non-esterified fatty acids (NEFA) into circulation. The liver takes up NEFA and either oxidizes them completely for energy, partially oxidizes them into ketone bodies such as beta-hydroxybutyrate (BHBA), or re-esterifies them into triglycerides for export as very low density lipoprotein or storage within hepatocytes. Because the bovine liver has a limited capacity to export triglyceride, excessive NEFA flux frequently results in hepatic lipidosis, commonly termed fatty liver, which in turn impairs gluconeogenesis and immune cell function, creating a self-reinforcing cycle of poor intake and worsening metabolic status.

Energy Balance and Subclinical Ketosis

Subclinical ketosis, defined as blood BHBA concentrations between 1.2 and 2.9 millimoles per liter without overt clinical signs, affects a substantial proportion of cows in a typical herd during the first two weeks postpartum, according to widely cited prevalence studies. Even in its subclinical form, elevated BHBA is associated with increased risk of displaced abomasum, metritis, reduced first-service conception rate, and lower milk yield across the subsequent lactation, making it an economically significant condition despite the absence of visible

illness. Nutritional strategies to mitigate NEB center on maximizing dry matter intake during the close-up period through diets with adequate but not excessive energy density, and on avoiding over-conditioning of dry cows, since cows entering the dry period with a body condition score above 3.5 on a five-point scale are at substantially elevated risk of excessive fat mobilization and subsequent ketosis and fatty liver. Feeding a controlled-energy, high-fiber close-up diet rather than a high-starch diet has become standard practice, since it limits excessive body condition gain in late gestation while still supporting rumen papillae adaptation for the fresh diet to follow. Supplementation with rumen-protected choline has demonstrated consistent benefit in reducing liver triglyceride accumulation, because choline is a required component of phosphatidylcholine, the phospholipid coat necessary for very low density lipoprotein assembly and hepatic triglyceride export. Glucogenic supplements such as propylene glycol, sodium propionate, or glycerol are commonly drenched or top-dressed in the first days after calving to spare glucose for lactose synthesis and to suppress ketogenesis.

Calcium Homeostasis and Hypocalcemia

Parturition triggers an abrupt calcium demand for colostrum synthesis, which can require the equivalent of the entire extracellular calcium pool within twenty-four hours of calving. Clinical hypocalcemia, or milk fever, occurs when the cow's homeostatic mechanisms, principally parathyroid hormone-mediated bone resorption and intestinal absorption, fail to compensate quickly enough, and it remains most prevalent in older, higher-producing cows. Subclinical hypocalcemia, defined as serum calcium below 8.0 milligrams per deciliter without recumbency, is now recognized as more economically important than the clinical form because of its high prevalence and its association with reduced smooth muscle contractility, which predisposes affected cows to retained placenta, displaced abomasum, and uterine prolapse. The dominant nutritional strategy to prevent hypocalcemia is manipulation of the dietary cation-anion difference (DCAD) during the close-up period.

Dietary Cation-Anion Difference Strategies

DCAD is calculated as the milliequivalent balance of dietary sodium and potassium (cations) minus chloride and sulfur (anions), and lowering this value through inclusion of anionic salts such as ammonium chloride, ammonium sulfate, or commercial anionic products induces a mild, controlled metabolic acidosis in the close-up cow. This acidotic state primes the parathyroid hormone receptor system and increases bone and intestinal calcium mobilization capacity before the calcium demand of calving actually arrives, so that the cow's homeostatic response is already upregulated at parturition. Target DCAD values for close-up diets typically range from negative 100 to negative 150 milliequivalents per kilogram of dry matter, and urine pH monitoring, with a target range of 6.0 to 6.5 for Holsteins, is used on farm to verify that the diet is achieving the intended degree of acidification without inducing excessive acidosis, which can itself depress intake. Adequate dietary magnesium, typically 0.35 to 0.4 percent of dry matter, is essential for this strategy to succeed because magnesium is a required cofactor for parathyroid hormone receptor signaling in bone and kidney tissue.

Rumen Adaptation and Feeding Management

Beyond energy and mineral metabolism, the transition period requires the rumen epithelium and microbial population to shift from a lower-starch dry cow diet to the higher-starch, higher-fermentable-carbohydrate lactation diet. Rumen papillae, which are responsible for volatile fatty acid absorption, require two to four weeks of exposure to fermentable substrate to increase in surface area sufficiently to handle the fresh cow diet without excessive volatile fatty acid accumulation and subacute ruminal acidosis. Step-up feeding programs that gradually increase the proportion of concentrate over the first two to three weeks of lactation, combined with adequate physically effective fiber to maintain rumination and salivary buffering, are essential to prevent acidosis during this adaptation window. Group housing strategies that minimize competition at the feed bunk, adequate bunk space of at least 75 centimeters per cow, and minimizing pen moves and social disruption during the close-up

and fresh periods further support consistent intake, since stress-induced cortisol release exacerbates both intake depression and immune suppression at a time when the cow is already immunocompromised.

Practical Recommendations for the Feeding Program

A well-designed transition program integrates these physiological principles into distinct feeding groups: a far-off dry group fed a high-fiber, moderate-energy diet to limit body condition gain; a close-up group, ideally managed for a minimum of 21 days, fed a negative DCAD diet with controlled energy density and adequate protein, typically 14 to 15 percent crude protein, to support colostrum synthesis and prepare rumen and calcium metabolism for lactation; and a fresh cow group monitored closely for intake, rumination time, and metabolic markers such as blood BHBA and NEFA, with targeted intervention protocols for animals identified as high risk. Routine monitoring of a representative sample of fresh cows, typically 10 to 12 days in milk, using cow-side BHBA meters allows herds to quantify subclinical ketosis prevalence and adjust the feeding program proactively rather than reactively. The economic case for this level of investment is strong: the downstream costs of a single case of clinical ketosis or displaced abomasum, including treatment, discarded milk, reduced fertility, and elevated culling risk, typically far exceed the marginal cost of a well-formulated close-up diet and routine metabolic monitoring program.

Monitoring, Record-Keeping, and Continuous Improvement

Sustained success in transition cow management depends not only on diet formulation but on systematic monitoring and record-keeping that allows the herd to detect drift in performance before it manifests as a spike in clinical disease. Beyond individual cow-side BHBA testing, herd-level metrics including the incidence of displaced abomasum, retained placenta rate, days to first breeding, and pregnancy rate by 150 days in milk serve as lagging indicators that reflect the cumulative success or failure of the transition program over the preceding weeks. Because many transition disorders share overlapping risk factors, a single case of displaced abomasum, for instance, should prompt review not only of that individual cow's history but of broader herd-level trends in dry matter intake, body condition score distribution at dry-off, and DCAD compliance, since isolated cases frequently signal a systemic issue affecting a wider cohort of at-risk cows rather than an isolated individual event. Regular forage testing is equally important, since the DCAD calculation and overall diet formulation depend on accurate mineral analysis of forage sources, and unrecognized shifts in forage potassium concentration, a common occurrence across cutting or field within the same farm, can silently undermine an otherwise well-designed close-up diet. Many herds now incorporate routine total mixed ration particle size analysis andorts, or refusal, monitoring as additional tools to verify that the diet delivered to the pen matches the diet as formulated, since sorting behavior, in which cows selectively consume the more palatable, higher-energy fraction of the ration while leaving long-stem fiber in the bunk, can materially alter the effective nutrient intake relative to the intended formulation, particularly during the vulnerable adaptation window of the first two weeks of lactation. Collaboration between the herd veterinarian, nutritionist, and farm management team, meeting on a regular cadence to review these metrics jointly, has been repeatedly identified in extension and applied research literature as a defining characteristic of herds that achieve consistently low transition disease incidence, underscoring that transition management is ultimately a systems-level discipline rather than a single dietary formula applied in isolation.

Conclusion

The transition period remains the pivotal determinant of a dairy cow's health, milk yield, and reproductive success for the entire lactation that follows. Because negative energy balance and the calcium demand of parturition are essentially unavoidable, the goal of transition nutrition is not to eliminate these physiological challenges but to manage their magnitude and duration through appropriate energy density, DCAD manipulation, choline and glucogenic

supplementation, and careful attention to intake-limiting factors such as bunk space, group stability, and body condition at dry-off. A nutritionally sound transition program, grounded in an understanding of hepatic lipid metabolism, calcium homeostasis, and rumen adaptation, remains one of the highest-return investments available to the modern dairy enterprise.

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