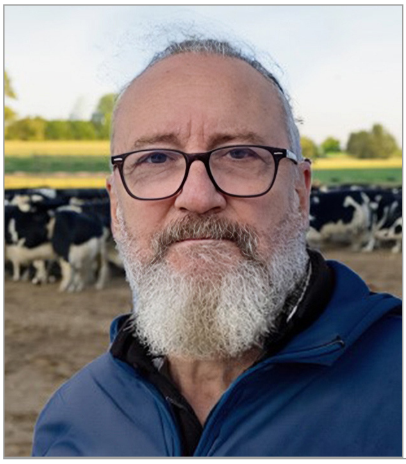

DDGS AS A POSTBIOTIC FEED INGREDIENT IN DAIRY COWS

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Intestinal barrier integrity plays a central role in the health and productivity of lactating dairy cows, particularly during periods of heat stress and metabolic strain. Distillers dried grains with solubles (DDGS), traditionally valued for their protein and energy contribution, also deliver fermentation-derived bioactive compounds. These components position DDGS as a potential postbiotic feed ingredient, with implications for gut permeability, immune activation, and oxidative stress regulation in dairy nutrition.

Intestinal barrier integrity is a key determinant of productivity and health in lactating dairy cows because it governs the balance between nutrient absorption and immune exclusion. The intestinal epithelium must permit efficient uptake of water, electrolytes, and nutrients while preventing translocation of luminal antigens, toxins, and microbial products into circulation. When barrier function is compromised, the resulting increase in intestinal permeability, commonly referred to as leaky gut syndrome, is associated with immune activation, oxidative stress, reduced feed efficiency, and lower milk yield, particularly during periods of heat stress and early lactation.

Distillers dried grains with solubles (DDGS) are widely used in dairy rations due to their favorable protein, energy, and phosphorus content, yet their effects on gut function are inconsistently described. This variability reflects the complex nature of DDGS, which concentrate not only nutrients but also fermentation-derived microbial residues, unsaturated lipids, fiber fractions, and, in some cases, mycotoxins from the parent grain. We recently proposed that DDGS can be viewed as a functional food postbiotic because they supply non-living, microbial-derived bioactive components generated during fermentation and processing rather than live microorganisms. This framework encourages evaluation of DDGS beyond nutrient supply and toward their potential effects on immune signaling, microbial ecology, and intestinal barrier function.

Specifically, this article links DDGS composition to the epithelial mechanisms that maintain or disrupt barrier integrity using the tight junction model illustrated in the accompanying figure.

TIGHT JUNCTION ARCHITECTURE AND BARRIER FUNCTION

The tight junction model illustrated in the accompanying figure below shows how epithelial barrier selectivity is maintained under homeostatic conditions and how oxidative and inflammatory stress can shift the system toward increased permeability. Tight junctions seal the apical lateral membranes of adjacent epithelial cells and collectively form the zonula occludens, a gasket-like structure that defines the paracellular pathway. Under normal conditions, this pathway permits controlled passage of water and small ions while restricting larger solutes and luminal macromolecules.

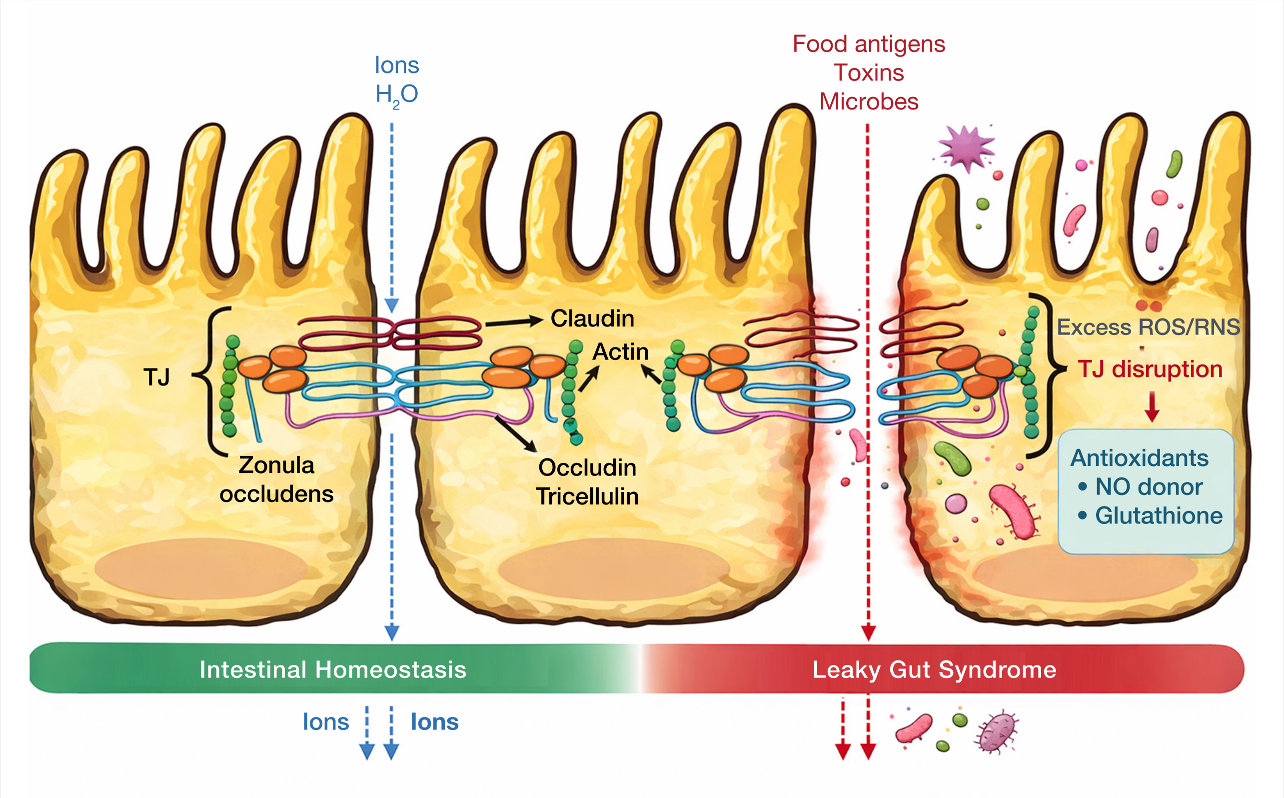
Barrier selectivity is governed primarily by transmembrane tight junction proteins. Claudins are the principal pore-forming components and determine ion selectivity and paracellular permeability based on isoform expression and junctional organization. Occludin contributes to junctional stability and stress responsiveness by regulating tight junction assembly and maintenance, while tricellulin is concentrated at tricellular junctions where three epithelial cells meet, sealing structurally vulnerable sites that would otherwise permit macromolecule leakage.

These proteins are anchored intracellularly to the actin cytoskeleton through adaptor proteins of the zonula occludens family. This cytoskeletal linkage allows tight junctions to respond dynamically to mechanical strain and intracellular signaling but also renders barrier integrity sensitive to cytoskeletal contraction or disorganization.

TRANSITION FROM INTESTINAL HOMEOSTASIS TO LEAKY GUT SYNDROME

Leaky gut syndrome emerges when oxidative and inflammatory stress disrupt tight junction structure and regulation. Excess reactive oxygen species and reactive nitrogen species (ROS and RNS) are central drivers of this transition. Oxidative and nitrosative stress alter signaling pathways that regulate tight junction protein trafficking, leading to internalization or redistribution of claudins and occludin away from the apical membrane and weakening their anchorage to actin. Concurrently, stress-induced cytoskeletal contraction widens paracellular spaces and compromises junctional cohesion.

Disruption of tricellulin localization further increases permeability at tricellular contacts, disproportionately facilitating passage of larger luminal antigens. Once barrier integrity is reduced, food antigens, toxins, and microbial products translocate across the epithelium and activate mucosal immune cells, amplifying inflammatory signaling and ROS production. This self-reinforcing cycle sustains tight junction disruption and perpetuates the leaky gut state.



Antioxidant systems modulate susceptibility of the epithelial barrier to oxidative stress. Glutathione serves as a primary intracellular antioxidant that neutralizes reactive species and protects tight junction proteins from oxidative modification, supporting maintenance of junctional localization and structure. Nitric oxide signaling contributes to epithelial perfusion and cellular communication under physiological conditions, but dysregulated nitric oxide metabolism promotes RNS accumulation and barrier dysfunction. Nutritional strategies that sustain antioxidant capacity therefore influence whether oxidative stress reaches a threshold sufficient to disrupt tight junction integrity.

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DDGS contain fermentation-derived residues from yeast and microbes generated during ethanol production, including cell wall-associated polysaccharides and other microbial-associated molecular patterns capable of interacting with host immune pathways and microbial communities. Although DDGS do not deliver live microbes and therefore are not probiotics, they can act as postbiotic-like inputs by supplying microbial-derived bioactive components that influence host physiology.

In dairy cows, fermentation products from Saccharomyces cerevisiae have been shown to stabilize microbial ecosystems and attenuate inflammatory responses during rumen and gut stress challenges. These findings support the biological plausibility that fermentation-derived bioactives can modulate microbial dynamics in ways that indirectly support barrier stability. However, DDGS differ from standardized fermentation products in that their composition varies substantially with processing history, storage conditions, and grain quality. As a result, the postbiotic hypothesis for DDGS is strongest when ingredient quality is consistent and does not introduce competing oxidative or toxic stress.

QUALITY-DEPENDENT RISK FACTORS LINKING DDGS TO BARRIER DISRUPTION

The potential barrier risks associated with DDGS are primarily quality dependent and map directly onto the oxidative mechanisms depicted in the tight junction model. High unsaturated lipid content can increase oxidative load when DDGS are heat damaged or poorly stored, promoting lipid peroxidation and epithelial oxidative stress. Oxidized lipids increase the likelihood of tight junction protein redistribution and cytoskeletal disruption.

Mycotoxins represent a second major risk factor. Because DDGS can concentrate mycotoxins from the parent grain, compounds such as deoxynivalenol may be present at biologically relevant levels. Deoxynivalenol is a well-documented epithelial stressor and tight junction disruptor, particularly under concurrent stress conditions such as heat stress or dietary acidosis. Dietary context further modulates risk, as highly fermentable diets and subacute ruminal acidosis shift microbial fermentation and inflammatory signaling, increasing susceptibility to permeability disturbances.

QUALITY AND INCLUSION BOUNDARIES FOR POSTBIOTIC PLAUSIBILITY

The postbiotic interpretation of DDGS depends on whether fermentation-derived residues are delivered without introducing oxidative or toxic stress sufficient to override barrier-supportive mechanisms. Lipid oxidation provides one functional boundary. Peroxide value serves as a practical indicator, with low values consistent with minimal oxidative burden and values approaching or exceeding approximately 20 mEq O_2/kg fat indicating substantial oxidation that reduces the likelihood of postbiotic-like effects.

Mycotoxin contribution represents a second boundary. When DDGS inclusion materially elevates total ration mycotoxin concentrations toward established guidance levels for dairy cattle, barrier disruption mechanisms are likely to dominate, making a postbiotic interpretation biologically implausible.

Postbiotic plausibility also depends on inclusion rate. Because DDGS are not standardized fermentation products, sufficient dietary inclusion is required to deliver a consistent quantity of fermentation-derived residues. In lactating cows consuming


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25 to 30 kg of dry matter per day, inclusion rates of approximately 10 to 20 percent of diet dry matter, corresponding to roughly 3 to 6 kg per cow per day, represent the most plausible range for DDGS to exert postbiotic-like effects when quality is high.

CONCLUSION

Leaky gut syndrome in dairy cows reflects disruption of tight junction protein organization, cytoskeletal anchoring, and redox balance. Claudins regulate selective paracellular permeability, occludin supports junctional stability and stress responsiveness, and tricellulin seals tricellular sites that otherwise permit macromolecule leakage. Excess ROS and RNS disrupt these proteins and their linkage to actin, widening the paracellular space and enabling translocation of luminal antigens, toxins, and microbes.

Within this mechanistic framework, DDGS should be regarded as a conditional functional ingredient in dairy diets. When sourced and handled to minimize oxidative damage and mycotoxin burden, and when included at appropriate rates in balanced rations that support antioxidant capacity, DDGS may contribute to microbial and immune stability consistent with a postbiotic framework. When these conditions are not met, oxidative and inflammatory pressures are more likely to dominate and promote the tight junction disruption processes associated with leaky gut syndrome.

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