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Commentary

Subclinical Mastitis in Dairy Cows by Managing Rumen Microbial Culture and Metabolites

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DESCRIPTION

One of the major illnesses that cause significant economic losses in the global dairy sector is Bovine Mastitis (BM). Due to great concealment and a lengthy latency period, Subclinical Mastitis (SCM), which makes up 90%-95% of BM in practical dairy cow production, is far more common than Clinical Mastitis (CM). Additionally, dairy cows with SCM in the herd are highly prone to infect other healthy people, leading to the development of secondary illnesses in the herd. While the milk and udders of cows with SCM may not show obvious alterations in appearance, harm is done to the immune system, normal metabolic processes, and lactation performance.

Therefore, the way to prevent future inflammatory aggravation is to execute the essential prevention and control measures during the SCM stage. One of the very contagious illnesses in dairy cows, Subclinical Mastitis (SCM), has a high incidence and no outward signs of clinical illness. Mastitis and the gut microbiota are intimately connected. A prebiotic fibre called inulin helps the host's immune system and intestinal microbial communities. However, it is still unclear how dietary inulin affects the rumen's internal environment. The goal of the current investigation was to determine whether inulin could reduce SCM symptoms in dairy cows by altering the ruminal bacterial and metabolite profiles.

Milk's Somatic Cell Counts (SCC) were decreased while milk output, protein, and lactose were all boosted by inulin. Interleukin-6 (IL-6), IL-8, Tumour Necrosis Factor (TNF), and Malondialdehyde (MDA) concentrations in serum were reduced, whereas IL-4 and Superoxide Dismutase (SOD) concentrations were elevated. In the rumen, inulin lowered NH3-N while increasing the concentrations of propionate, butyrate, and Lactic Acid (LA). Numerous beneficial commensal bacteria, such as Muribaculaceae and *Bifidobacterium*, as well as metabolites related to energy and amino acid metabolism, such as melibiose and l-glutamate, were increased. These included bacteria that produce propionate and butyrate, such as Prevotella and Butyrivibrio. The downregulation of lipid proinflammatory metabolites such ceramide and 17-phenyl-trinor-prostaglandin E2 was accompanied by the reduction of a number of proinflammatory microorganisms, including *Streptococcus* and *Escherichia-Shigella*. The 300 g/day inulin group in the current study had the best response, according to the aforementioned indications. Overall, adding inulin to the diet could reduce inflammatory reactions in cows with SCM by enhancing the rumen's internal environment. It has been established that dairy cows' gut microbiomes and mastitis are related. Subclinical mastitis may be cured by controlling the rumen bacteria profile (SCM). Inulin supplementation in cow diets with SCM may boost levels of amino acids and energy metabolism while also increasing the number of Short-Chain Fatty Acid (SCFA) producing bacteria and beneficial commensal bacteria in the rumen. On the other hand, proinflammatory ruminal microorganisms and metabolites were less common. This shows that enhancing the rumen's internal environment with inulin supplementation may be able to reduce inflammatory reactions in dairy cows during SCM, enhancing lactation efficiency and milk quality. Our findings offer a theoretical foundation for SCM regulatory strategies in dairy cows. According to studies, the gut microbiota may have an impact on the development of systemic inflammatory disorders. Composition of the microbiota may be regulated by intestinal immune responses brought on by commensal populations. The danger of pathogen infection, harmful pathogen overgrowth, and inflammatory disorders rises when the natural microbial ecosystem is destroyed. Some experimenters gave sterile mice mastitis-like symptoms by transplanting the faces of cows with the disease into their intestines.

In contrast to healthy cows, our prior research showed that cows with SCM and CM had much lower commensal populations, including bacteria that produce Short-Chain Fatty Acids (SCFA), as compared to healthy cows. Additionally, the drop in SCFAs brought on by intestinal dysbacteriosis may lead the blood-milk barrier to become even more permeable, aggravating mastitis. The endogenous entero-mammary route of intestinal bacteria is the basis for their effect on mastitis. Contrary to monogastric mammals, dairy cows have the biggest microbial community in their rumen, which also happens to be the primary organ for the production of SCFAs. In order to ease SCM in dairy cows, we therefore tried to modify the rumen interior environment by dietary management.

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