



HYPERSENSITIVITY TO GLUTEN AND LACTOSE

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ABSTRACT

This thesis explores the pathomorphology behind increased sensitivity to gluten and lactose intolerance. Gluten sensitivity and lactose intolerance are increasingly prevalent, impacting the quality of life for many individuals. Gluten sensitivity, a spectrum of disorders including celiac disease and non-celiac gluten sensitivity (NCGS), affects individuals' immune systems, leading to intestinal inflammation. Lactose intolerance, caused by the inability to properly digest lactose due to lactase deficiency, manifests primarily through gastrointestinal symptoms. This paper will discuss the underlying mechanisms of these conditions, focusing on their pathological basis, diagnostic challenges, and potential treatment options.

Introduction. Gluten sensitivity and lactose intolerance are common causes of gastrointestinal discomfort, with their prevalence rising globally. Gluten sensitivity is a broad term that includes both celiac disease, an autoimmune condition, and NCGS, a condition with symptoms similar to celiac disease but without the associated autoimmunity. Lactose intolerance, on the other hand, is characterized by the inability to digest lactose, the sugar found in dairy products, due to low lactase enzyme levels. Both conditions can lead to symptoms such as bloating, diarrhea, and abdominal pain, which impact patients' quality of life. Understanding the pathological processes underlying these conditions is essential for better diagnosis and treatment.

Methods. A systematic review of current literature was conducted to explore the pathomorphology, pathophysiology, diagnostic criteria, and management strategies for gluten sensitivity and lactose intolerance. The search included peer-reviewed articles from pathology, gastroenterology, and nutrition journals, using keywords such as "gluten sensitivity," "celiac disease," "non-celiac gluten sensitivity," "lactose intolerance," "pathomorphology," "pathophysiology," and "intestinal inflammation." The articles were critically analyzed to provide an overview of the underlying mechanisms of these conditions and their implications for patient care.

Results. Gluten Sensitivity. Celiac disease is an autoimmune disorder triggered by the ingestion of gluten in genetically predisposed individuals. The interaction between gluten and the immune system results in damage to the small intestine, characterized by villous atrophy, crypt hyperplasia, and increased intraepithelial lymphocytes. The primary immune response

is mediated by T-cells, leading to chronic inflammation and malabsorption. NCGS presents with similar symptoms, but without the autoimmune markers or intestinal damage seen in celiac disease. The exact mechanism behind NCGS remains unclear, though it is thought to involve a combination of immune activation and gut barrier dysfunction.

Lactose Intolerance. Lactose intolerance occurs due to lactase deficiency, either genetically programmed (primary lactose intolerance) or secondary to conditions that damage the small intestine, such as celiac disease or Crohn's disease. Lactase is responsible for breaking down lactose into glucose and galactose. In the absence of adequate lactase, undigested lactose ferments in the colon, producing gases and leading to symptoms such as bloating, diarrhea, and abdominal pain. Primary lactose intolerance is particularly prevalent in certain populations, including individuals of African, Asian, and Native American descent, where lactase activity declines after weaning.

Discussion. The rising prevalence of gluten sensitivity and lactose intolerance highlights the importance of understanding the underlying pathological processes. In celiac disease, the immune-mediated destruction of the small intestinal mucosa leads to malabsorption and systemic complications if untreated. While the exact etiology of NCGS is unclear, its increasing diagnosis suggests the need for further research into immune and non-immune mechanisms. Lactose intolerance, although often benign, can significantly impact dietary choices and gastrointestinal health.

Diagnosing these conditions can be challenging due to the overlap in symptoms. Celiac disease is diagnosed through serological tests and confirmed by biopsy, while NCGS is a diagnosis of exclusion. Lactose intolerance can be diagnosed through a lactose tolerance test, hydrogen breath test, or genetic testing.

Treatment for gluten sensitivity requires a strict gluten-free diet, particularly in cases of celiac disease, to prevent intestinal damage. For lactose intolerance, dietary modifications such as limiting dairy intake or using lactase supplements can alleviate symptoms. Emerging therapies, including enzyme supplements and microbiome-based interventions, may provide alternative treatment options in the future.

Conclusion. Gluten sensitivity and lactose intolerance are significant contributors to gastrointestinal distress and warrant further attention in clinical and research settings. While the mechanisms of celiac disease and lactose intolerance are well understood, NCGS remains an area for further study. Improved diagnostic criteria and treatments are needed to help manage these conditions and improve patient outcomes. Understanding the pathology behind gluten and lactose intolerance will enhance the ability of clinicians to diagnose and treat these conditions effectively.

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