Review of: "The Role of Ferroptosis in Inflammatory Bowel Disease: Mechanisms and Therapeutic Implications"

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Potential competing interests: No potential competing interests to declare.

I have thoroughly reviewed the manuscript entitled 'The Role of Ferroptosis in Inflammatory Bowel Disease: Mechanisms and Therapeutic Implications," which investigates the role of ferroptosis in the pathology of Inflammatory Bowel Disease (IBD), encompassing both Crohn's disease and ulcerative colitis. The manuscript provides a comprehensive review of the current understanding of ferroptosis and its distinctive mechanisms, including the roles of GPx4, Nrf2-HO-1 pathways, and iron metabolism within the context of IBD. Moreover, it elaborates on the dual nature of iron in intestinal health and disease and discusses the implications of ferroptosis for intestinal epithelial cell death, barrier function, and immune response, underscoring its potential as a novel therapeutic target. Strengths: Relevance and Timeliness: The topic is highly relevant and timely, given the increasing incidence of IBD worldwide and the pressing need for novel therapeutic strategies. Comprehensive Review: The manuscript offers a thorough review of ferroptosis and its mechanisms, contributing valuable insights into the complex interplay between metabolic, inflammatory, and cell death pathways in IBD. Novel Perspective: By focusing on ferroptosis, the manuscript provides a unique perspective on the potential for new therapeutic approaches, highlighting the importance of nutrition, genetics, and immunity in intestinal health and disease. Areas for Improvement: Mechanism Clarification: While the manuscript effectively outlines the roles of various pathways and metabolic processes, a deeper and more detailed explanation of how these mechanisms specifically contribute to the pathology of IBD would enhance the reader's understanding. Empirical Evidence: The manuscript would benefit from a more detailed discussion of empirical studies supporting the role of ferroptosis in IBD, including both animal models and, if available, human studies. Therapeutic Potential: While the potential for targeting ferroptosis in IBD therapy is highlighted, a more critical analysis of the challenges and limitations faced in translating these findings into clinical practice is needed. This includes addressing current gaps in research and potential side effects of manipulating ferroptosis pathways. Conclusion: This manuscript provides significant insights into the role of ferroptosis in IBD and presents a compelling case for its potential as a therapeutic target. However, to fully realize the manuscript's impact and relevance, I recommend addressing the above areas for improvement. Specifically, elaborating on the mechanisms, providing more empirical evidence, and critically analyzing the therapeutic potential and challenges would greatly enhance the manuscript's contribution to the field. I look forward to seeing the revisions and believe that this manuscript has the potential to make a valuable contribution to the understanding and treatment of IBD.