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Unraveling autophagy-related pathogenesis in active ulcerative colitis: A bioinformatics approach

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Abstract

In this editorial, we provide commentary on the study by Gong *et al*. In this original research article, Gong *et al* employed a bioinformatics approach to investigate the involvement of autophagy in active ulcerative colitis (UC). Through differential gene expression analysis, they identified 58 differentially expressed autophagy-related genes in UC patients compared to healthy controls. Notably, HSPA5, CASP1, SERPINA1, CX3CL1, and BAG3, were found to be up-regulated in active UC patients, suggesting their significance as core autophagy-related targets. Enrichment analysis unveiled associations with crucial signaling pathways and diseases such as middle cerebral artery occlusion and glomerulonephritis. Moreover, immune cell infiltration analysis revealed notable differences in immune cell composition between UC patients and healthy controls. These findings offer valuable insights into the role of autophagy in UC pathogenesis and potential therapeutic targets.

Key Words: Ulcerative colitis; Autophagy; Bioinformatics; Pathogenesis; Therapeutic targets

Core Tip: The study by Gong *et al* highlights HSPA5, CASP1, SERPINA1, CX3CL1, and BAG3 as core autophagy-related targets upregulated in active ulcerative colitis (UC) patients. Enrichment analysis underscores the involvement of autophagy in key signaling pathways and diseases associated with UC. Immune cell infiltration analysis emphasizes the importance of immune dysregulation in UC progression. These findings provide crucial insights into the molecular mechanisms underlying UC pathogenesis and may guide the development of personalized treatment strategies for UC patients.

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INTRODUCTION

Autophagy dysregulation is a key factor in the pathogenesis of active ulcerative colitis (UC)[1]. Given its role, targeting autophagy pathways represents a crucial therapeutic approach for active UC[1]. Other strategies include dietary interventions that modulate the gut microbiota and short-chain fatty acids[2]. In this context, Gong *et al*[3] have provided valuable insights into the complex pathogenesis of active UC, highlighting the significance of autophagy-related mechanisms. Their bioinformatics analyses identified key autophagy-related genes and pathways that are dysregulated in active UC, shedding light on potential therapeutic targets and disease predictors[3]. Autophagy plays a vital role in maintaining intestinal homeostasis and protecting against inflammatory bowel diseases such as UC[4]. By facilitating the degradation of intracellular pathogens and damaged organelles, autophagy helps regulate inflammation and prevent tissue damage in the gut[4]. Enhancing autophagy has been proposed as a therapeutic approach for UC, as it can reduce inflammation and promote mucosal healing[5,6]. Therefore, targeting autophagy pathways may offer promising strategies for treating UC and other inflammatory bowel diseases. Gong *et al*'s study identified critical autophagy-related targets, including HSPA5, CASP1, SERPINA1, CX3CL1, and BAG3, which are upregulated in active UC patients[3]. These targets are associated with essential signaling pathways such as autophagy in animals and lipid and atherosclerosis pathways. Additionally, the study revealed a significant link between autophagy-related pathogenesis in active UC and other diseases like middle cerebral artery occlusion and glomerulonephritis[3]. These findings underscore the pivotal role of autophagy in UC pathogenesis, influencing immune cell infiltration and signaling pathways.

DETAILED MECHANISMS AND THERAPEUTIC IMPLICATIONS

Inhibiting autophagy has emerged as a potential strategy to mitigate UC damage. Autophagy is crucial for maintaining cellular homeostasis by degrading and recycling damaged cellular components. In the context of UC, dysregulated autophagy can lead to excessive inflammation and tissue damage. Studies have shown that autophagy helps to regulate immune responses and maintain gut integrity, suggesting that enhancing autophagy could be beneficial in UC treatment [4,7]. The identification of autophagy-related genes such as HSPA5 and CASP1 highlights the intricate relationship between autophagy and inflammation in UC. HSPA5, also known as glucose-regulated protein 78, is involved in the unfolded protein response and helps protect cells from stress-induced damage. CASP1, a caspase involved in the inflammasome pathway, plays a role in the maturation of pro-inflammatory cytokines such as interleukin-1 β . Targeting these genes could provide new avenues for therapeutic intervention[3]. Dietary interventions that influence autophagy can also modulate the gut microbiota and short-chain fatty acids (SCFAs), which are crucial for gut health. Konjac glucomannan polysaccharide and inulin oligosaccharide have been shown to ameliorate colitis by restoring gut microbiota balance and enhancing SCFA production[2]. These findings suggest that combining autophagy-targeting therapies with dietary modifications could have synergistic effects in UC treatment. Gong *et al*[3] identified several autophagy-related therapeutic targets that are upregulated in UC. These include BAG3, a co-chaperone that regulates autophagy and apoptosis, and CX3CL1, a chemokine involved in immune cell migration. The upregulation of these targets in UC patients suggests their potential as biomarkers for disease severity and therapeutic response. Furthermore, targeting these molecules could help modulate the autophagic response and reduce inflammation in UC[3].

FUTURE DIRECTIONS AND CLINICAL IMPLICATIONS

The study by Gong *et al*[3] provides a foundation for developing novel therapies that target autophagy-related pathways in UC. The identification of key genes and signaling pathways involved in autophagy opens new avenues for drug development. For instance, pharmacological agents that enhance autophagy, such as Lonicerin, which targets EZH2 to

alleviate UC by autophagy-mediated NOD-, LRR- and pyrin domain-containing protein 3 inflammasome inactivation, have shown promise in preclinical studies[5]. Further research is needed to understand the molecular mechanisms underlying autophagy dysregulation in UC. Investigating the interactions between autophagy-related genes and key signaling molecules will provide insights into the complex network of pathways involved in UC pathogenesis. This knowledge will be crucial for developing targeted therapies that can precisely modulate autophagy and improve clinical outcomes for UC patients[7]. Clinical trials are essential to validate the efficacy and safety of new autophagy-targeting therapies. Studies focusing on the pharmacokinetics, pharmacodynamics, and therapeutic potential of autophagy modulators will help translate these findings into clinical practice. Additionally, identifying biomarkers that predict patient response to autophagy-targeting therapies will enable personalized treatment approaches[6].

CONCLUSION

In summary, the dysregulation of autophagy plays a critical role in the pathogenesis of active UC. Targeting autophagy pathways offers a promising therapeutic strategy for this chronic inflammatory disease. The study by Gong *et al*[3] provides valuable insights into autophagy-related mechanisms and identifies potential therapeutic targets for UC. Further research is essential to understand the molecular mechanisms involved and to develop novel therapies that can improve clinical outcomes for UC patients. The integration of autophagy-targeting treatments with dietary and other interventions holds potential for a comprehensive approach to managing UC.

FOOTNOTES

Author contributions: Hao WR and Cheng CY wrote the initial paper; Liu JC and Cheng TH revised the paper. All authors have read and approved the final manuscript. Hao WR and Cheng CY provided the initial draft of the editorial, synthesizing the key findings and implications of Gong *et al*'s study. Liu JC and Cheng TH played crucial roles in revising the manuscript, ensuring it accurately reflects the significance of the findings and their implications for future research and clinical practice. Liu JC contributed his expertise in interpreting the relevance of the bioinformatics approach and the identified autophagy-related genes. Cheng TH provided valuable insights into the broader context of the study, guiding the narrative to emphasize the clinical importance of the findings. Both Liu and Cheng were essential in refining the editorial's structure, integrating feedback from co-authors, and finalizing the manuscript for submission. Their substantial and indispensable contributions to the manuscript's development and their guidance throughout the revision process justify their roles as co-corresponding authors.

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