Research Progress on the NLRP3 Inflammasome in Digestive System Diseases

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Abstract Digestive system diseases seriously threaten human physical and mental health, with their incidence remaining persistently high. The NLRP3 inflammasome is a core multiprotein complex of the innate immune system, regulating the maturation and release of pro-inflammatory factors such as IL-1β and IL-18. The NLRP3 inflammasome, as a key innate immune sensor, plays a significant role in the occurrence and development of various digestive system diseases. This paper reviewed the research progress on the NLRP3 inflammasome in digestive system diseases, and explored the relationship between the NLRP3 inflammasome and these diseases, aiming to provide a theoretical basis for the treatment of digestive system diseases.

Key words NLRP3; Inflammasome; Digestive system diseases

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Digestive system diseases refer to conditions where the normal anatomical integrity or physiological function of the digestive system is compromised due to structural abnormalities, functional disorders, pathogen infections, or immune imbalances[1]. Furthermore, these diseases exhibit a high lifetime prevalence in the general population, and with the acceleration of population aging, the incidence and mortality of digestive system diseases are also rising[2]. Therefore, great importance should be attached to digestive system health, and proactive preventive measures should be taken to reduce the occurrence of digestive diseases. The NLRP3 inflammasome, as a core pattern recognition receptor complex of the innate immune system, plays a key role in recognizing pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs). Upon activation, it mediates Caspase-1-dependent maturation of inflammatory factors and pyroptosis, extensively participating in the onset and progression of various inflammatory diseases[3]. Abnormal activation of the NLRP3 inflammasome is closely associated with the development of multiple digestive system diseases[4]. In-depth research into the mechanism of the NLRP3 inflammasome in digestive system diseases holds significant importance for therapeutic strategies targeting these conditions.

Overview of the NLRP3 Inflammasome

The NLRP3 inflammasome is a key multiprotein complex that mediates intracellular inflammatory responses. Its functional activity is directly determined by its structural composition and dynamic assembly process. It is primarily composed of the NLRP3 protein, apoptosis-associated speck-like protein containing a CARD (ASC), and cysteine aspartic acid specific protease-1 (caspase-1). NLRP3 belongs to the NOD-like receptor (nucleotide-binding oligomerization domain-like receptor, NLR) family and is composed of an N-terminal Pyrin Domain (PYD), a central NACHT domain, and a C-terminal LRR domain. It interacts with the PYD of the adaptor protein ASC through its own PYD domain, and ASC further recruits caspase-1 via its CARD domain, and finally, an active NLRP3 inflammasome complex is assembled[5]. When cells are stimulated, NLRP3 recognizes various danger signals through its leucine-rich repeat (LRR) domain. It then interacts with the ASC protein via its PYD domain, and subsequently recruits procaspase-1 through the CARD domain to form a complete inflammasome complex[6].

Activation of the NLRP3 Inflammasome

The activation mechanism of the NLRP3 inflammasome as a critical component of the innate immune system plays a central role in host defense against pathogens and inflammatory responses triggered by cellular damage. The activation process of the NLRP3 inflammasome is highly complex and finely regulated, typically involving two stages of signals: the priming signal and the activation signal. The priming signal is triggered by Toll-like receptors (TLRs), which promote the transcriptional expression of NLRP3 and pro-IL-1β via the NF-κB pathway. The activation signal, on the other hand, is triggered by various DAMPs or PAMPs, including ATP, reactive oxygen species (ROS), and mitochondrial DNA[7]. These signals lead to K^+ efflux, mitochondrial dysfunction, cathepsin release, and lysosomal rupture, thereby facilitating the oligomerization of NLRP3 and the assembly of the inflammasome[8]. Among these, K^+ efflux serves as a common mechanism for NLRP3 activation. Various NLRP3 agonists disrupt cell membrane permeability to K^+, reducing intracellular K^+ concentration

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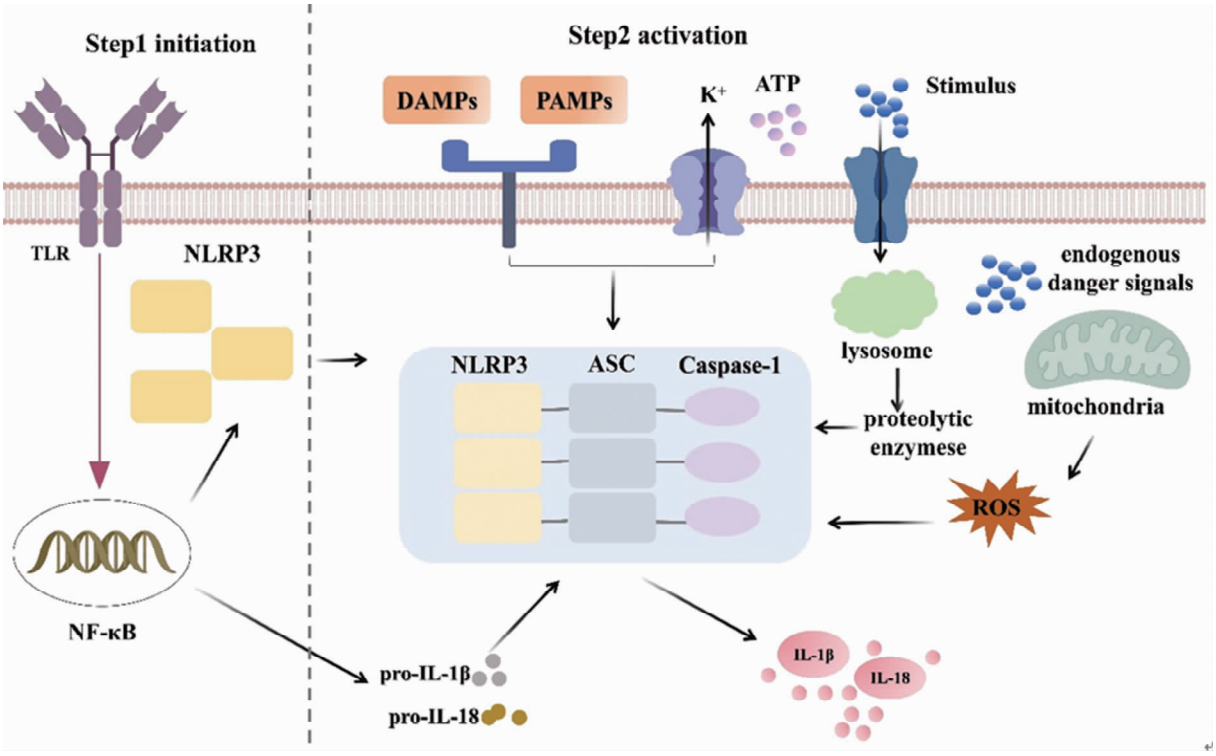
and thereby activating the inflammasome[8]. Additionally, increased generation of reactive oxygen species (ROS) due to mitochondrial dysfunction and the release of cathepsins from lysosomal rupture are critical upstream signals for NLRP3 activation[9].



Classical conditions for NLRP3 inflammasome oligomerization and activation include the action of ATP (adenosine triphosphate), DAMPs and PAMPs, lysosomal damage, cathepsin release, K^+ efflux, and mitochondrial ROS damage.

Fig. 1 The priming step of NLRP3 mediated by TLRs promoting NF-κB activation and inducing NLRP3 expression

NLRP3 Inflammasome and Digestive System Diseases

In recent years, with the deepening research on the regulatory mechanisms and signaling pathways of NLRP3 inflammasome activation, the NLRP3 inflammasome has been implicated in numerous digestive tract diseases, including liver injury, liver cancer, acute pancreatitis, inflammatory bowel disease, gastric ulcers, and gastric cancer. An in-depth analysis of the association between the NLRP3 inflammasome and various digestive system diseases not only helps elucidate the molecular mechanisms underlying disease pathogenesis and progression, but also provides a theoretical basis for developing novel therapeutic targets.

NLRP3 inflammasome and non-alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is a progressive liver condition characterized by ectopic fat accumulation in the liver caused by factors other than alcohol[10]. Activation of the NLRP3 inflammasome in hepatocytes is associated with the pathogenesis of liver disease[11]. In the canonical pathway of NLRP3 inflammasome activation, the NF-κB signaling pathway is first activated, creating conditions for NLRP3 inflammasome activation. Subsequently, NLRP3 assembles with ASC and Caspase-1 to form an active complex, mediating the cleavage and maturation of the pro-inflammatory cytokines IL-1β and IL-18. This process triggers local liver inflammation, exacerbating hepatocellular steatosis and injury[12]. Wang *et al.*[13] observed that in a mouse NAFLD model induced by an MCD diet, the levels of NLRP3-related proteins were significantly elevated in the livers of wild-type (WT) mice. It was accompanied by increases in triglycerides, serum alanine aminotransferase (ALT), and aspartate aminotransferase (AST), indicating hepatocellular injury. In contrast, NLRP3 gene knockout (NLRP3 −/−) mice exhibited only half the hepatic lipid accumulation of WT mice, along with significantly reduced levels of inflammatory factors and diminished severity of hepatocellular damage. MCC950, a targeted inhibitor of the NLRP3 inflammasome, directly binds to the Walker B motif in the NACHT domain of the NLRP3 protein, inhibiting ATP hydrolysis and thereby suppressing NLRP3 activation and inflammasome assembly[14]. Studies have shown that MCC950 treatment has the potential to alleviate liver inflammation and improve liver fibrosis, mitigating pathological damage in NAFLD[15]. Thus, inhibiting NLRP3 inflammasome assembly can effectively ameliorate the progression of NAFLD.

NLRP3 inflammasome and liver cancer

Hepatocellular carcinoma (HCC) is a malignant tumor originating from liver cells. Chronic persistent inflammation can cause hepatocellular damage, leading to liver fibrosis and cirrhosis, and ultimately progressing to HCC[16]. The activation of the NLRP3 inflammasome is closely associated with tumorigenesis. As a core component of the inflammatory response, the inflammasome plays a

dual role in tumor development. Lee's study found that NLRP3 gene knockout in HCC cells led to increased surface expression of MICA/B, reducing tumor growth and metastasis[17]. Li *et al.*[18] discovered that anisodamine inhibited NLRP3 inflammasome activation, further suppressing caspase-1 activation and the release of IL-1β and IL-18, ultimately inhibiting the occurrence of HCC. In targeted therapy research, when sorafenib was combined with the NLRP3 inflammasome-targeted inhibitor MCC950, antitumor activity increased, accompanied by an expansion of tumor necrotic areas and a reduction in proliferating cell nuclear antigen (PCNA)-positive cells[19]. Dai *et al.*[20] found that Shuanghua decoction promoted ROS release, and thus activated the NLRP3 inflammasome while inhibiting the NF-κB signaling pathway. These responses further led to the upregulation of NLRP3, pro-IL-1β, and cleaved-caspase-1 expression, enhanced LPS + ATP-induced NLRP3 inflammasome activation and ultimately inhibited HCC. These findings reveal the multifaceted role of the NLRP3 inflammasome in HCC, providing new insights for developing therapeutic strategies targeting HCC.

NLRP3 inflammasome and acute pancreatitis

Acute pancreatitis (AP) is characterized by inflammation of the exocrine pancreas, primarily caused by gallstones, alcohol consumption, and hypertriglyceridemia[21]. In AP, the NLRP3 inflammasome serves as a key mediator of pancreatic inflammatory responses. Its activation is closely associated with macrophage infiltration and activation, and it regulates downstream caspase-1 expression via the MyD88/NF-κB pathway, exacerbating pancreatic edema, cytokine storms, and tissue damage. Therefore, the NLRP3 inflammasome is a critical driver of pathological progression in AP[22]. Wen *et al.*[23] induced a mouse AP model by intraperitoneal injection of cerulein. In AP mice, both the NLRP3 inflammasome and caspase-1 were activated, and as the expression of NLRP3 inflammasome components increased, the levels of IL-1β and IL-18 also rose correspondingly. Matthias Sendler[24] reported that NLRP3 activation mediated IL-18 secretion, induced an increase in Th2 cell numbers, and promoted neutrophil infiltration and T-cell activation, thereby exacerbating pancreatic inflammatory damage. In contrast, NLRP3 gene knockout or intervention with its inhibitor MCC950 significantly reduced T-cell activation and neutrophil infiltration, suppressed Th2-mediated immune responses, and alleviated AP. In severe acute pancreatitis (SAP), activation of the NLRP3 inflammasome can synergize with the TLR4-MAPK/NF-κB pathway to promote the release of pro-inflammatory factors such as IL-1β and IL-6, exacerbating pathological damage in pancreatic and ileal tissues[25]. Therefore, intervention strategies targeting the NLRP3 inflammasome provide a new direction for the treatment of AP.

NLRP3 inflammasome and inflammatory bowel disease

Inflammatory bowel disease (IBD), primarily comprising Crohn's Disease (CD) and Ulcerative Colitis (UC), is characterized by hyperactive immune responses leading to abnormal inflammation in the digestive system and intestinal tissues[26]. Liao *et al.*[27] revealed that abnormal activation of NLRP3 could lead to disruption of the intestinal mucosal barrier and excessive inflammatory responses. VSIG4 expression was downregulated in IBD patients and negatively correlated with NLRP3 inflammasome activity, and overexpression of VSIG4 could inhibit NLRP3 activation and the expression of pyroptosis-related proteins in M1-type macrophages. Studies have found that in the DSS-induced colitis model, the NLRP3 inflammasome serves as a core inflammatory regulatory mechanism. Gene knockout studies showed that deletion of NLRP3, ASC or caspase-1 could completely block the production of IL-1β induced by DSS. *In-vivo* experiments further revealed that, compared with wild-type mice, NLRP3 knockout mice exhibited significantly alleviated colitis damage and reduced levels of pro-inflammatory cytokines after DSS induction[28]. The NLRP3 inflammasome plays a driving role in the pathogenesis of UC. When intestinal immune homeostasis is disrupted, various PAMPs and DAMPs can trigger the activation of the NLRP3 inflammasome, initiating the Caspase-1-dependent pyroptosis process. It further leads to permeable swelling and lysis of macrophages, releasing large amounts of lactate dehydrogenase and inflammatory factors such as IL-1β and IL-18[29]. As a key molecular target for the treatment of ulcerative colitis, the NLRP3 inflammasome opens new research directions for IBD therapy.

NLRP3 inflammasome and colorectal cancer

Colorectal cancer (CRC) is a major global health issue. Persistent intestinal inflammatory responses can lead to genetic damage in colon cells, and the frequent renewal of damaged intestinal mucosal cells disrupts intestinal homeostasis, ultimately potentially inducing CRC[30]. Studies have shown that high expression of NLRP3 is associated with poor prognosis in CRC patients. The activation of the NLRP3 inflammasome not only affects the state of tumor cells, but also directly influences the function of immune cells in the tumor microenvironment and the secretion of inflammatory factors[31]. Liang *et al.*[32] found that ROS generated by abnormal metabolism in CRC cells activated the NLRP3 inflammasome, thereby exacerbating the proliferation, invasion, and metastasis of CRC cells. Abnormal activation of the NLRP3 inflammasome played a central role in maintaining the malignant phenotype of CRC cells and constructing an immunosuppressive tumor microenvironment. The study by Zhang *et al.*[33] confirmed that inhibiting the NF-κB pathway could block NLRP3 inflammasome activation, reduce apoptosis and pyroptosis in CRC cells, and arrest the cell cycle at the G2/M phase to inhibit cell growth and migration. It could also reverse the immunosuppressive tumor microenvironment. Therefore, the NLRP3 inflammasome can be used as a potential target for CRC treatment.

NLRP3 inflammasome and gastric ulcer

Gastric ulcer (GU) is a common chronic ulcerative disease of the digestive system, characterized by localized erosion and necrotic lesions in the gastric mucosal and submucosal layers, representing a significant global health burden[34]. Key etiological factors of GU include *Helicobacter pylori* (Hp) infection, long-term use of non-steroidal anti-inflammatory drugs (NSAIDs), and ethanol consumption. In ethanol-induced GU models, ethanol stimulation leads to gastric mucosal damage and extensive edema, reduced glandular structure, significant inflammatory cell infiltration into the mucosal layer, and upregulation of NLRP3, ASC, and caspase-1 expression in gastric tissues, triggering inflammasome

activation[35]. Multiple natural products can inhibit the NLRP3 inflammasome to exert anti-GU effects. Liang *et al.*[36] found that CRP significantly alleviated ethanol-induced GU by targeting the NLRP3/ASC/Caspase-1 signaling axis, thereby producing a gastric mucosal protective effect. Therefore, inhibiting the NLRP3/ASC/Caspase-1 signaling axis may play an important role in the treatment of GU.

NLRP3 inflammasome and gastric cancer

Gastric cancer (GC) is a highly prevalent and life-threatening gastrointestinal malignancy worldwide[37]. Chronic inflammation is a major contributing factor to GC. NLRP3, as a key inflammasome component, plays a critical role in triggering inflammation. Studies have shown that the NLRP3 inflammasome is localized in gastric cancer tissues. The expression levels of NLRP3 and ASC, as well as the activity of caspase-1, are significantly upregulated in both GC cells and GC tissues. Moreover, the activation of the NLRP3 inflammasome is closely associated with the NF-κB pathway. In LPS-treated mouse GC models and human gastric cancer BGC-823 cells, inhibition of the NF-κB pathway suppresses NLRP3 inflammasome activation, thereby curbing inflammatory responses and tumor proliferation[38]. The study by Zhang *et al.*[39] revealed that CagA could induce intracellular ROS generation, activate the NLRP3 inflammasome, upregulate the expression of NLRP3, caspase-1, and ASC proteins, and promote the release of IL-1β and IL-18, thereby facilitating pyroptosis in gastric cancer cells. Therefore, blocking NLRP3 inflammasome activation could attenuate the pro-migration and pro-invasion effects of CagA. These findings further clarify the role of the NLRP3 inflammasome in promoting GC progression, providing a theoretical basis for targeting the NLRP3 signaling pathway to intervene in GC.

Conclusions and Prospects

The NLRP3 inflammasome, as a key molecular complex in innate immune responses, has been extensively reported in *in-vivo* experiments, *in-vitro* models, and clinical studies for its association with digestive system diseases, establishing it as a core target in the molecular mechanisms of these conditions. Understanding the functional differences of the NLRP3 inflammasome in various digestive system diseases, elucidating its regulatory relationship with disease onset and progression and targeting the inhibition of the NLRP3 inflammasome or its downstream inflammatory cytokines such as IL-1β and IL-18 can provide new insights for the treatment of digestive system diseases including non-alcoholic fatty liver disease, liver cancer, acute pancreatitis, inflammatory bowel disease, colorectal cancer, gastric ulcer, and gastric cancer. It also lays a theoretical foundation for developing novel drugs that target the regulation of inflammatory pathways.

References

[1] WANG Q, GUO F, JIN Y, *et al.* Applications of human organoids in the personalized treatment for digestive diseases[J]. Signal Transduct Target Ther, 2022, 7(1): 336.

[2] RICCIARDIELLO L. Digestive diseases: Big burden, low funding? Results of the new United European Gastroenterology White Book on digestive diseases[J]. United European Gastroenterol J, 2022, 10(7): 627 – 628.

[3] LUO L, WANG F, XU X, *et al.* STAT3 promotes NLRP3 inflammasome activation by mediating NLRP3 mitochondrial translocation[J]. Exp Mol Med, 2024, 56(9): 1980 – 1990.

[4] QIANG R, LI Y, DAI X, *et al.* NLRP3 inflammasome in digestive diseases: From mechanism to therapy[J]. Front Immunol, 2022, 13: 978190.

[5] REN W, SUN Y, ZHAO L, *et al.* NLRP3 inflammasome and its role in autoimmune diseases: A promising therapeutic target[J]. Biomed Pharmacother, 2024, 175: 116679.

[6] GAIDT MM, EBERT TS, CHAUHAN D, *et al.* The DNA inflammasome in human myeloid cells is initiated by a STING-cell death program upstream of NLRP3[J]. CELL, 2017, 171(5): 1110.

[7] JO EK, KIM JK, SHIN DM, *et al.* Molecular mechanisms regulating NLRP3 inflammasome activation[J]. Cell Mol Immunol, 2016, 13(2): 148 – 159.

[8] GAIDT MM, HORNUNG V. The NLRP3 inflammasome renders cell death pro-inflammatory[J]. Journal of Molecular Biology, 2018, 430(2): 133 – 141.

[9] LUO T, ZHOU X, QIN M, *et al.* Corilagin restrains NLRP3 inflammasome activation and pyroptosis through the ROS / TXNIP / NLRP3 pathway to prevent inflammation[J]. Oxidative Medicine and Cellular Longevity, 2022: 2022.

[10] LI SJ, LIU AB, YU YY, *et al.* The role and mechanism of pyroptosis and potential therapeutic targets in non-alcoholic fatty liver disease (NAFLD)[J]. Front Cell Dev Biol, 2024, 12: 1407738.

[11] SZABO G, CSAK T. Inflammasomes in liver diseases[J]. J Hepatol, 2012, 57(3): 642 – 654.

[12] WANG Q, OU Y, HU G, *et al.* Naringenin attenuates non-alcoholic fatty liver disease by down-regulating the NLRP3/NF-kappaB pathway in mice[J]. Br J Pharmacol, 2020, 177(8): 1806 – 1821.

[13] LI JL, DING YN. Inhibitory Effect of Erhuang Jieyu Tang on Cell proliferation and MMP-2 Expression in HepG2 Cells[J]. Journal of Hubei University of Medicine, 2012, 31(1): 46 – 48.

[14] COLL RC, HILL JR, DAY CJ, *et al.* MCC950 directly targets the NLRP3 ATP-hydrolysis motif for inflammasome inhibition[J]. Nat Chem Biol, 2019, 15(6): 556 – 559.

[15] QI J, YAN X, LI L, *et al.* CXCL5 promotes lipotoxicity of hepatocytes through upregulating NLRP3/Caspase-1/IL-1beta signaling in Kupffer cells and exacerbates nonalcoholic steatohepatitis in mice[J]. Int Immunopharmacol, 2023, 123: 110752.

[16] STAUFFER JK, SCARZELLO AJ, JIANG Q, *et al.* Chronic inflammation, immune escape, and oncogenesis in the liver: A unique neighborhood for novel intersections[J]. Hepatology, 2012, 56(4): 1567 – 1574.

[17] LEE H H, KIM D, JUNG J, *et al.* NLRP3 deficiency in hepatocellular carcinoma enhances surveillance of NK-92 through a modulation of MICA/B[J]. Int J Mol Sci, 2021, 22(17).

[18] LI P, LIU Y, HE Q. Anisodamine suppressed the growth of hepatocellular carcinoma cells, induced apoptosis and regulated the levels of inflammatory factors by inhibiting NLRP3 inflammasome activation[J]. Drug Des Devel Ther, 2020, 14: 1609 – 1620.

[19] TUTUSAUS A, SANDUZZI-ZAMPARELLI M, BOIX L, *et al.* Induction of the inflammasome pathway by tyrosine kinase inhibitors provides an actionable therapeutic target for hepatocellular carcinoma[J]. Cancers

(Basel), 2024, 16(8).
[20] DAI B, FAN M, HUANG X, *et al.* Shuanghua decoction exerts anticancer activity by activating NLRP3 inflammasome via ROS and inhibiting NF-kappaB signaling in hepatocellular carcinoma cells[J]. Phytomedicine, 2022, 103: 154249.
[21] JABBOUR E, KANTARJIAN H. Chronic myeloid leukemia: A review [J]. JAMA, 2025, 333(18): 1618 – 1629.
[22] HOU C, ZHU X, SHI C, *et al.* Iguratimod (T-614) attenuates severe acute pancreatitis by inhibiting the NLRP3 inflammasome and NF-kappaB pathway[J]. Biomed Pharmacother, 2019, 119: 109455.
[23] WEN Y, HAN C, LIU T, *et al.* Chaiqin Chengqi Decoction alleviates severity of acute pancreatitis via inhibition of TLR4 and NLRP3 inflammasome: Identification of bioactive ingredients via pharmacological subnetwork analysis and experimental validation[J]. Phytomedicine, 2020, 79: 153328.
[24] SENDLER M, van den BRANDT C, GLAUBITZ J, *et al.* NLRP3 inflammasome regulates development of systemic inflammatory response and compensatory anti-inflammatory response syndromes in mice with acute pancreatitis[J]. Gastroenterology, 2020, 158(1): 253 – 269.
[25] ZHANG ZY, GUO XL, LIU JT, *et al.* Conjugated bile acids alleviate acute pancreatitis through inhibition of TGR5 and NLRP3 mediated inflammation[J]. J Transl Med, 2024, 22(1): 1124.
[26] ZHEN Y, ZHANG H. NLRP3 inflammasome and inflammatory bowel disease[J]. Front Immunol, 2019, 10: 276.
[27] LIAO Y, DENG C, WANG X. VSIG4 ameliorates intestinal inflammation through inhibiting macrophages NLRP3 inflammasome and pyroptosis [J]. Tissue Cell, 2024, 86: 102285.
[28] BAUER C, DUEWELL P, MAYER C, *et al.* Colitis induced in mice with dextran sulfate sodium (DSS) is mediated by the NLRP3 inflammasome[J]. Gut, 2010, 59(9): 1192 – 1199.
[29] LARABI A, BARNICH N, NGUYEN H. New insights into the interplay between autophagy, gut microbiota and inflammatory responses in IBD [J]. Autophagy, 2020, 16(1): 38 – 51.
[30] MOHAPATRA B, LAVUDI K, KOKKANTI R R, *et al.* Regulation of NLRP3/TRIM family signaling in gut inflammation and colorectal cancer [J]. Biochim Biophys Acta Rev Cancer, 2025, 1880(2): 189271.
[31] LI S, WANG X, HONG L, *et al.* NLRP3 promotes the proliferation and metastasis of colorectal cancer by regulating the S6K1-GLI1 signaling pathway[J]. J Cancer, 2025, 16(2): 521 – 532.
[32] LIANG L, SUN W, WEI X, *et al.* Oxymatrine suppresses colorectal cancer progression by inhibiting NLRP3 inflammasome activation through mitophagy induction *in vitro* and *in vivo*[J]. Phytother Res, 2023, 37 (8): 3342 – 3362.
[33] ZHANG G, YAO Y, ZHANG Z, *et al.* Regulation of NLRP3 inflammasome and Caspase-3/4/11 by 2', 4'-dihydroxychalcone contributes to anti-colorectal cancer[J]. Phytomedicine, 2024, 135: 156194.
[34] GONG H, ZHAO N, ZHU C, *et al.* Treatment of gastric ulcer, traditional Chinese medicine may be a better choice[J]. J Ethnopharmacol, 2024, 324: 117793.
[35] SELIM H M, NEGM W A, HAWWAL M F, *et al.* Fucoidan mitigates gastric ulcer injury through managing inflammation, oxidative stress, and NLRP3-mediated pyroptosis [J]. Int Immunopharmacol, 2023, 120: 110335.
[36] LIANG H, LIANG Y, WU L, *et al.* Pretreatment with *Citrus reticulata* 'Chachi' polysaccharide alleviates alcohol-induced gastric ulcer by inhibiting NLRP3/ASC/Caspase-1 and Nrf2/HO-1 signaling pathways [J]. Nutrients, 2025, 17(13).
[37] Erratum: Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries[J]. CA Cancer J Clin, 2020, 70(4): 313.
[38] CASTANO-RODRIGUEZ N, KAAKOUSH NO, MITCHELL HM. Pattern-recognition receptors and gastric cancer[J]. Front Immunol, 2014, 5: 336.
[39] ZHANG X, LI C, CHEN D, *et al.* H. pylori CagA activates the NLRP3 inflammasome to promote gastric cancer cell migration and invasion[J]. Inflamm Res, 2022, 71(1): 141 – 155.

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References

[1] State Council. Plan for bioeconomic development during the 14th Five-Year Plan[Z]. Beijing: General Office of the State Council, 2022.
[2] Ministry of Education. National standards for teaching quality in undergraduate majors in general higher education institutions (Bioengineering category)[S]. Beijing: Higher Education Press, 2018.
[3] MyCOS Institute. 2025 Chinese undergraduate employment report[R]. Beijing: Social Sciences Literature Press, 2025. (in Chinese).
[4] WU JR. Teaching reform of bioengineering specialty in colleges and universities: Analysis and discussion from the development of bio-industry and the employment of college students[J]. Education Forum, 2014 (23): 42 – 44. (in Chinese).
[5] 51job. Research report on fresh graduates in 2025[R]. Shanghai: 51job, 2025. (in Chinese).
[6] ZOU WT, WANG YJ, CAO Z. Application of virtual simulation technology in bioengineering experiment teaching[J]. University, 2025(5): 95 – 98.
[7] FENG LH, TIAN H, YIN YS. Exploration and practice of MOOC construction based on VR technology and intelligent teaching mode based on MOOC[J]. China Educational Technology & Equipment, 2025(8): 14 – 17.
[8] CAI Y, CHANG F, XIA M, *et al.* Exploration and practice for personnel training mode of biological engineering based on education training plan for excellent engineers[J]. Farm Products Processing, 2022(1): 94 – 101. (in Chinese).
[9] SUN BC, HU S. Evaluation mechanism of employment impact of college graduates: Model construction and policy effect[J]. Jiaoyu Yanjiu, 2025, 6(545): 61 – 73. (in Chinese).
[10] HUANG HS. Wuxi City: "Training + employment" to build a "career express" for college graduates[J]. China Employment, 2025(7): 56 – 57. (in Chinese).

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