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Review Article

Unravelling the Role of the NLRP3 Inflammasome in Cardiovascular Diseases

Prashu Yadav

Phd Scholar, NIILM University, India.
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E-mail Id:
Prashuyadav18@gmail.com
Orcid Id:
https://orcid.org/0009-0003-9709-9739
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A B S T R A C T

Cardiovascular disorders (CVDs) continue to be a major global health concern, requiring ongoing research into new treatment approaches. Emerging research has highlighted the intricate role of inflammation, particularly mediated by the NLRP3 inflammasome, in the pathogenesis of CVDs. This review provides an overview of the NLRP3 inflammasome, its function, regulation, and implications in health and disease. In order to clarify the underlying mechanisms, we address its role in cardiovascular diseases such as atherosclerosis, myocardial infarction, heart failure, and hypertension. Additionally, we explore the therapeutic potential of targeting the NLRP3 inflammasome in CVDs, highlighting ongoing preclinical studies and clinical trials. Despite the promise of NLRP3 inhibition, several challenges exist, including achieving specificity, identifying biomarkers, and ensuring long-term safety. We propose future research directions, emphasising the need for personalised approaches, combination therapies, and translational research to realise the full therapeutic potential of targeting the NLRP3 inflammasome in combating CVDs.

Keywords: NLRP3, Cardiovascular Diseases, Noncanonical Inflammasome

Introduction

As a major cause of death globally, cardiovascular diseases (CVDs) place a heavy strain on both individuals and healthcare systems. Although smoking, high blood pressure, and high cholesterol are well-established risk factors, new research is illuminating the complex role that inflammation plays in the onset and progression of CVDs. Among the key players in this inflammatory cascade is the NLRP3 inflammasome, a multiprotein complex implicated in various cardiovascular pathologies.[1] Understanding the nuances of its involvement in CVDs holds promise for the development of novel therapeutic strategies.

NLRP3 Inflammasome: A Brief Overview

In the intricate world of immunology, the NLRP3 inflammasome stands out as a central orchestrator of inflammation, playing a pivotal role in the body's defence mechanisms and contributing significantly to the pathogenesis of various diseases. The NOD-like receptor protein 3 (NLRP3), adaptor protein ASC, and effector molecule caspase-1 make up this molecular complex, which acts as a sentinel for signals of cellular stress and danger. It sets off a series of events that result in the production of pro-inflammatory cytokines such as interleukin-1β (IL-1β) and interleukin-18 (IL-18).[2] Understanding the intricacies

of the NLRP3 inflammasome has far-reaching implications, offering insights into both health and disease.

Function and Regulation

The NLRP3 inflammasome acts as a guardian of cellular homeostasis, sensing a diverse array of danger signals ranging from microbial pathogens and toxins to endogenous danger-associated molecular patterns (DAMPs). Upon activation, NLRP3 undergoes conformational changes, facilitating its interaction with ASC and subsequent recruitment of procaspase-1, leading to its autocatalytic cleavage and activation. Active caspase-1 then cleaves pro-IL-1β and pro-IL-18 into their mature forms, initiating a potent inflammatory response.[3,4]

The regulation of NLRP3 activation is a subject of intense research. Various stimuli, including ATP, reactive oxygen species (ROS), mitochondrial dysfunction, and lysosomal damage, have been implicated in triggering NLRP3 activation. Moreover, a delicate balance of inhibitory and activating signals fine-tunes the NLRP3 inflammasome activity, ensuring appropriate responses to different stimuli and preventing aberrant inflammation.[3]

Implications in Health and Disease

While the NLRP3 inflammasome is essential for host defence against infections and tissue repair, dysregulated NLRP3 activation has been implicated in the pathogenesis of numerous inflammatory and autoimmune diseases. Inflammatory bowel disease, Alzheimer's disease, and atherosclerosis are among the illnesses where chronic activation of the NLRP3 inflammasome has been found to contribute to tissue destruction and the advancement of the disease. Conversely, deficiencies in NLRP3 activation have been linked to increased susceptibility to certain infections.[4]

Targeting the NLRP3 Inflammasome

Given its central role in inflammation and disease, the NLRP3 inflammasome has emerged as an attractive therapeutic target. Strategies aimed at modulating NLRP3 activation include small molecule inhibitors, monoclonal antibodies, and gene-editing techniques. Several preclinical studies and clinical trials are underway to evaluate the efficacy and safety of NLRP3 inhibitors in various inflammatory conditions, holding promise for the development of novel therapeutic interventions.[5]The multiprotein complex known as the NLRP3 inflammasome is a crucial component of the innate immune system and is involved in both initiating and escalating inflammatory responses. Procaspase-1, the adaptor protein ASC (apoptosis-associated speck-like protein containing a CARD), and the NLRP3 sensor protein comprise the NLRP3 inflammasome, which serves as a molecular platform for the activation of inflammatory caspases. Environmental and metabolic stressors, as well as a variety of endogenous danger signals, such as pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs), activate it.

The activation route of the NLRP3 inflammasome involves two distinct signals. NLRP3 and pro-IL-1β are transcriptionally upregulated in response to the priming signal, which primes the cell for more activation. Toll-like receptors (TLRs) and cytokine receptors are examples of pattern recognition receptors (PRRs) that often mediate it. The second signal, triggered by various stimuli such as microbial components, environmental toxins, or cellular stressors, leads to the assembly of the NLRP3 inflammasome complex, culminating in the proteolytic cleavage of pro-caspase-1 into its active form. Activated caspase-1 then cleaves pro-inflammatory cytokines pro-IL-1β and pro-IL-18 into their biologically active forms, IL-1β and IL-18, respectively, promoting inflammation and pyroptotic cell death.[4–6]

Notably, the NLRP3 inflammasome exhibits a remarkable degree of versatility in its activation, responding to a wide range of stimuli through diverse signalling pathways. These stimuli include but are not limited to extracellular ATP, pore-forming toxins, crystalline and particulate matter, mitochondrial dysfunction, and metabolic dysregulation. The ability of the NLRP3 inflammasome to sense such a diverse array of danger signals underscores its significance as a central mediator of inflammation and immune responses in various disease states.

Moreover, dysregulated NLRP3 inflammasome activation has been implicated in the pathogenesis of numerous inflammatory and autoimmune disorders, including but not limited to rheumatoid arthritis, inflammatory bowel disease, and neuroinflammatory diseases such as Alzheimer's disease and multiple sclerosis. In these conditions, aberrant NLRP3 activation contributes to chronic inflammation, tissue damage, and disease progression, highlighting its potential as a therapeutic target for intervention.

In the context of cardiovascular diseases, the NLRP3 inflammasome has emerged as a key player in driving inflammation and tissue damage within the cardiovascular system. Its activation in response to various endogenous danger signals, such as cholesterol crystals, oxidised LDL, and mitochondrial dysfunction, contributes to endothelial dysfunction, vascular inflammation, atherosclerotic plaque formation, myocardial injury, and adverse cardiac remodelling. Targeting the NLRP3 inflammasome pathway holds promise for the development of novel therapeutic strategies to combat cardiovascular diseases and improve patient outcomes.[5–7]

Thus, the NLRP3 inflammasome represents a pivotal node in the intricate network of innate immune signalling,

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orchestrating inflammatory responses in health and disease. Its versatile activation and involvement in a wide range of pathological conditions underscore its significance as a potential therapeutic target for intervention. Further elucidating the molecular mechanisms governing NLRP3 inflammasome activation and regulation will pave the way for the development of precision-targeted therapies for inflammatory and cardiovascular diseases. Figure 1 describes the Noncanonical Inflammasone Activation.

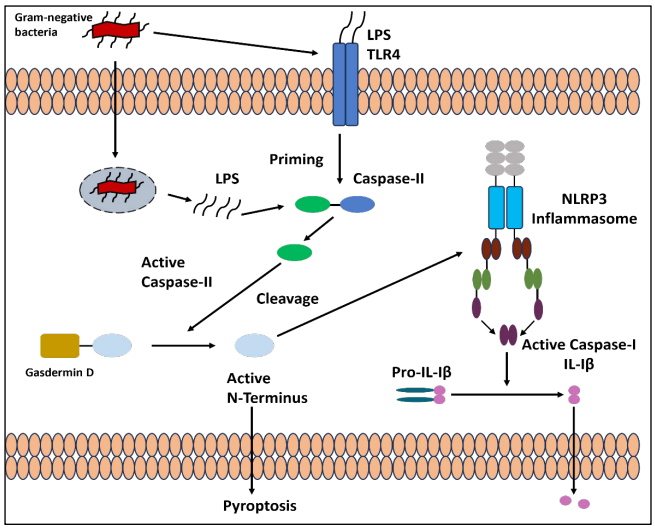

Figure 1.Noncanonical Inflammasome Activation

Noncanonical Inflammasome Activation

The process by which inflammasomes, which are multiprotein complexes involved in innate immune responses, are activated outside of the canonical pathway is known as noncanonical inflammasome activation. Inflammasomes are essential for identifying intracellular infections, danger signals, and environmental stressors. They also cause pyroptotic cell death and the release of pro-inflammatory cytokines like interleukin-1β (IL-1β) and interleukin-18 (IL-18).

Pathogen-associated molecular patterns (PAMPs) or danger-associated molecular patterns (DAMPs) are usually recognised by pattern recognition receptors (PRRs), such as NOD-like receptors (NLRs) or missing in melanoma 2 (AIM2)-like receptors, as part of the canonical inflammasome activation pathway. The PRR, the adaptor protein ASC (apoptosis-associated speck-like protein containing a caspase recruitment domain), and the effector enzyme caspase-1 come together to form the inflammasome complex as a result of this recognition.[8]

In contrast, noncanonical inflammasome activation occurs primarily in response to specific bacterial infections, particularly those involving gram-negative bacteria such as Escherichia coli, Salmonella typhimurium, and Legionella pneumophila. These bacteria possess lipopolysaccharide (LPS) in their outer membrane, which can directly activate caspase-11 (or its human orthologs, caspase-4 and caspase-5) in a cytosolic manner.

LPS causes caspase-11 to get activated, which sets off a series of events that culminate in pyroptosis, a highly inflammatory kind of programmed cell death. When caspase-11 is activated, gasdermin D is cleaved, releasing its N-terminal portion that creates holes in the cell membrane, causing osmotic swelling and ultimately cell lysis. This process facilitates the release of pro-inflammatory cytokines and danger signals, contributing to host defence against infection.

Noncanonical inflammasome activation can also induce the production and release of IL-1β and IL-18, although the mechanisms involved are less well-defined compared to the canonical pathway. Recent studies have suggested that noncanonical inflammasome activation may involve the activation of the NLRP3 inflammasome downstream of caspase-11 activation, as well as the involvement of other caspases such as caspase-8.

Overall, noncanonical inflammasome activation represents an important mechanism by which the immune system detects and responds to intracellular bacterial infections, contributing to host defence and inflammatory responses. Further research into the molecular mechanisms and regulation of this pathway may uncover new therapeutic targets for the treatment of infectious and inflammatory diseases.[9,10]

Nexus between NLRP3 Inflammasome and Cardiovascular Diseases

The pathophysiology of several CVDs, such as atherosclerosis, myocardial infarction (MI), heart failure, and hypertension, appears to be significantly influenced by the NLRP3 inflammasome, according to growing data. In atherosclerosis, the initial step in CVD development, NLRP3 activation in response to cholesterol crystals and oxidised LDL within the arterial wall triggers the release of IL-1β and IL-18, promoting endothelial dysfunction, leukocyte recruitment, and plaque formation. Moreover, NLRP3-mediated inflammation exacerbates plaque vulnerability, increasing the risk of plaque rupture and acute cardiovascular events such as MI and stroke.

Through the production of pro-inflammatory cytokines and the generation of pyroptosis, ischaemia injury in MI exacerbates myocardial damage by inducing NLRP3 activation within cardiac myocytes and infiltrating immune cells. Similar to this, persistent NLRP3 inflammasome activation in heart failure feeds the vicious cycle of inflammation and cardiac decompensation by causing detrimental cardiac remodelling, fibrosis, and dysfunction.

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Furthermore, emerging evidence suggests a role for the NLRP3 inflammasome in the pathogenesis of hypertension, with NLRP3 activation implicated in endothelial dysfunction, vascular remodelling, and renal injury. These findings underscore the broad impact of NLRP3-mediated inflammation on diverse aspects of cardiovascular health.[11]

In the context of atherosclerosis, the chronic inflammatory response orchestrated by the NLRP3 inflammasome is intricately involved in all stages of plaque development. Endothelial dysfunction, characterised by impaired vasodilation and increased vascular permeability, represents an early hallmark of atherosclerosis. Activation of NLRP3 within endothelial cells promotes the expression of adhesion molecules and chemokines, facilitating the recruitment and transmigration of monocytes into the subendothelial space. Once infiltrated, monocytes differentiate into macrophages and engulf modified lipoproteins, leading to the formation of foam cells, a key component of atherosclerotic plaques.

Moreover, NLRP3 activation within macrophages promotes the secretion of matrix metalloproteinases (MMPs) and pro-inflammatory cytokines, destabilising the fibrous cap of atherosclerotic plaques and increasing the risk of rupture and thrombosis. Interestingly, recent studies have highlighted the crosstalk between NLRP3-mediated inflammation and lipid metabolism, with NLRP3 activation exacerbating dyslipidemia and promoting the accumulation of lipids within atherosclerotic lesions.[12,13]

Ischaemic damage in the context of acute myocardial infarction (MI) sets off a strong inflammatory response that is typified by the migration of circulating immune cells to the infarcted myocardium and the activation of local cardiac cells. NLRP3 inflammasome activation within cardiac myocytes and infiltrating leukocytes promotes the production of pro-inflammatory cytokines, such as IL-1β and IL-18, exacerbating myocardial damage and impairing cardiac function. Additionally, NLRP3-mediated pyroptosis, a form of inflammatory cell death, further amplifies tissue injury and contributes to adverse remodelling of the infarcted heart.

Beyond the acute phase, chronic activation of the NLRP3 inflammasome has been implicated in the progression of heart failure, a complex clinical syndrome characterised by impaired cardiac function and neurohormonal dysregulation. In response to hemodynamic stress and neurohumoral activation, cardiac myocytes undergo hypertrophic remodelling, accompanied by fibrosis and chamber dilatation. Persistent NLRP3 activation within the failing heart promotes interstitial fibrosis and myocyte apoptosis, contributing to contractile dysfunction and progressive heart failure. Furthermore, NLRP3-mediated inflammation has been implicated in endothelial dysfunction and microvascular rarefaction, exacerbating myocardial ischaemia and impairing myocardial perfusion in the failing heart.

In the realm of hypertension, a chronic elevation in blood pressure exerts deleterious effects on the structure and function of the vasculature, predisposing individuals to end-organ damage, including myocardial hypertrophy, renal dysfunction, and stroke. Emerging evidence suggests a crucial role for the NLRP3 inflammasome in mediating vascular inflammation and endothelial dysfunction in hypertension. NLRP3 activation within endothelial cells promotes the expression of adhesion molecules and vasoconstrictors, impairing vasodilation and promoting vascular remodelling. Additionally, NLRP3-mediated inflammation contributes to renal injury and salt-sensitive hypertension, further exacerbating cardiovascular risk in hypertensive individuals.[14,15]

Overall, the intricate interplay between NLRP3-mediated inflammation and cardiovascular diseases underscores the multifaceted role of innate immunity in the pathogenesis of CVDs. Targeting the NLRP3 inflammasome holds promise for the development of innovative therapeutic strategies aimed at mitigating inflammation-driven cardiovascular damage and improving patient outcomes. However, further research is needed to unravel the complexities of NLRP3 signalling and its implications for cardiovascular health, paving the way for personalised approaches to CVD management.

Therapeutic Implications and Future Directions

Given the central role of the NLRP3 inflammasome in cardiovascular pathologies, targeting this inflammatory pathway holds promise for the development of novel therapeutic interventions. Several preclinical studies have demonstrated the efficacy of NLRP3 inhibitors, such as MCC950 and glyburide, in attenuating atherosclerosis, myocardial infarction, and heart failure in experimental models. Moreover, clinical trials evaluating the safety and efficacy of NLRP3 inhibitors in CVD patients are currently underway, offering hope for the translation of these findings into clinical practice.[16]

However, several challenges remain in harnessing the therapeutic potential of NLRP3 inhibition, including off-target effects, specificity of targeting, and the complex interplay between inflammation and tissue repair processes. Future research efforts should focus on elucidating the precise mechanisms underlying NLRP3 activation in different cardiovascular pathologies, as well as identifying novel therapeutic targets within the inflammasome signalling cascade.

While the potential of targeting the NLRP3 inflammasome for therapeutic intervention in cardiovascular diseases (CVDs) is promising, several important considerations and future directions merit attention.

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- **Selective Targeting of NLRP3:** One of the major challenges in developing NLRP3 inhibitors lies in achieving specificity without interfering with other inflammasome complexes or essential immune functions. Further research is needed to identify specific molecular targets within the NLRP3 signalling cascade that can be modulated to inhibit inflammasome activation selectively.
- **Combination Therapies:** Given the multifactorial nature of CVDs, combination therapies targeting multiple pathways, including inflammation, lipid metabolism, and vascular function, may offer synergistic benefits. Combinatorial approaches integrating NLRP3 inhibitors with existing pharmacotherapies, such as statins and antiplatelet agents, warrant investigation to determine their efficacy and safety profile.
- **Personalised Medicine:** The heterogeneity observed in CVDs necessitates a personalised approach to treatment. Biomarkers indicative of NLRP3 inflammasome activation and disease severity could aid in patient stratification and selection of appropriate therapeutic interventions. Moreover, genetic polymorphisms influencing NLRP3 activity may inform individualised treatment strategies tailored to the patient's genetic profile.
- **Exploring Alternative Therapeutic Modalities:** In addition to small molecule inhibitors, alternative therapeutic modalities targeting NLRP3 inflammasome activation are being explored. These include biologic agents such as monoclonal antibodies targeting IL-1β or IL-18, as well as gene therapy approaches aimed at modulating NLRP3 expression or activity in specific cell types or tissues.
- **Translational Research and Clinical Trials:** Translating preclinical findings into clinical practice requires rigorous evaluation of the safety and efficacy of NLRP3 inhibitors in human subjects. Large-scale randomised controlled trials are needed to assess the impact of NLRP3 inhibition on clinical endpoints, including cardiovascular events, mortality, and quality of life, across diverse patient populations.
- **Long-term Safety Profile:** Given the chronic nature of CVDs, long-term safety monitoring of NLRP3 inhibitors is paramount. Comprehensive assessment of potential adverse effects, including immunosuppression, infection risk, and effects on tissue repair and regeneration, is essential to ensure the overall benefit-risk profile of these agents.
- **Beyond Traditional Therapies:** In addition to pharmacological interventions, lifestyle modifications targeting inflammation, such as diet, exercise, and stress management, may complement conventional therapies for CVDs. Understanding the interplay between lifestyle factors and NLRP3-mediated inflammation could inform holistic approaches to disease management and prevention.

In conclusion, while the therapeutic targeting of the NLRP3 inflammasome holds considerable promise for the treatment of cardiovascular diseases, translating these advances into clinical practice requires a multidisciplinary approach encompassing basic science, translational research, and clinical trials. By addressing the aforementioned challenges and leveraging emerging technologies and therapeutic modalities, we can harness the full potential of NLRP3 inhibition to combat the global burden of cardiovascular diseases and improve patient outcomes.[16]

Conclusion

The NLRP3 inflammasome represents a critical link between inflammation and cardiovascular diseases, orchestrating a cascade of inflammatory responses that contribute to disease initiation and progression. Targeting NLRP3-mediated inflammation holds promise for the development of innovative therapeutic strategies aimed at mitigating the burden of CVDs and improving patient outcomes. However, further research is warranted to unravel the complexities of NLRP3 signalling and its implications for cardiovascular health.

The intricate interplay between the NLRP3 inflammasome and cardiovascular diseases underscores the pivotal role of innate immunity in the pathogenesis of CVDs. The NLRP3 inflammasome acts as a central mediator of inflammation and tissue damage within the cardiovascular system, contributing to atherosclerosis, myocardial infarction, heart failure, and hypertension through diverse mechanisms. Targeting the NLRP3 inflammasome pathway holds promise for the development of innovative therapeutic strategies aimed at mitigating inflammation-driven cardiovascular damage and improving patient outcomes.

However, harnessing the therapeutic potential of NLRP3 inhibition poses several challenges, including achieving specificity, identifying biomarkers, and ensuring long-term safety. Future research efforts should focus on elucidating the precise mechanisms underlying NLRP3 activation in different cardiovascular pathologies, exploring combination therapies, and advancing personalised medicine approaches tailored to individual patient profiles.

Furthermore, a thorough assessment of the effects of NLRP3 inhibitors on clinical endpoints must be conducted through extensive randomised controlled trials in order to translate preclinical findings into clinical practice. To guarantee the overall benefit-risk balance of these therapeutic medicines, thorough evaluation of potential side effects and long-term safety monitoring are crucial.

Beyond pharmacological interventions, lifestyle modifications targeting inflammation may complement

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conventional therapies for CVDs. By addressing these challenges and leveraging emerging technologies and therapeutic modalities, we can harness the full potential of NLRP3 inhibition to combat the global burden of cardiovascular diseases and improve patient outcomes.

Conflict of Interest: None

References

1. Caocao C, Juanjuan T, Jiaxin J, Yalong K, Haifang W. Advances in the role of NLRP3 inflammasome in cardiovascular diseases. MEDS Basic Med. 2024 Jan 12;2(1):15-23. [Google Scholar]
2. Stergiou IE, Tsironis C, Papadakos SP, Tsitsilonis OE, Dimopoulos MA, Theocharis S. Unraveling the role of the NLRP3 inflammasome in lymphoma: implications in pathogenesis and therapeutic strategies. Int J Mol Sci. 2024 Feb 17;25(4):2369. [PubMed] [Google Scholar]
3. Liu Y, Li X, Sun T, Li T, Li Q. Pyroptosis in myocardial ischemia/reperfusion and its therapeutic implications. Eur J Pharmacol. 2024;971:176464. [PubMed] [Google Scholar]
4. Del Buono MG, Bonaventura A, Vecchié A, Moroni F, Golino M, Bressi E, De Ponti R, Dentali F, Montone RA, Kron J, Lazzerini PE, Crea F, Abbate A. Pathogenic pathways and therapeutic targets of inflammation in heart diseases: a focus on interleukin-1. Eur J Clin Invest. 2024 Feb;54(2):e14110. [PubMed] [Google Scholar]
5. Yi YS. Roles of the caspase-11 non-canonical inflammasome in rheumatic diseases. Int J Mol Sci. 2024 Feb 8;25(4):2091. [PubMed] [Google Scholar]
6. Coudereau R, Bodinier M, Lukaszewicz AC, Py BF, Argaud L, Cour M, Bidar F, Cerrato E, Garnier L, Gossez M, Venet F, Monneret G. Persistent NLRP3 inflammasome activation is associated with delayed immunosuppression in septic patients. J Leukoc Biol. 2024;115(4):706-13. [PubMed] [Google Scholar]
7. Toldo S, Abbate A. The role of the NLRP3 inflammasome and pyroptosis in cardiovascular diseases. Nat Rev Cardiol. 2024 Apr;21(4):219-37. [PubMed] [Google Scholar]
8. Xu Z, Kombe Kombe AJ, Deng S, Zhang H, Wu S, Ruan J, Zhou Y, Jin T. NLRP inflammasomes in health and disease. Mol Biomed. 2024 Apr 22;5(1):14. [PubMed] [Google Scholar]
9. Toldo S, Abbate A. The NLRP3 inflammasome in acute myocardial infarction. Nat Rev Cardiol. 2018;15:203-14. [Google Scholar]
10. Jin Y, Fu J. Novel insights into the NLRP 3 inflammasome in atherosclerosis. J Am Heart Assoc. 2019 Jun 18;8(12):e012219. [PubMed] [Google Scholar]
11. Jiang C, Xie S, Yang G, Wang N. Spotlight on NLRP3 inflammasome: role in pathogenesis and therapies of atherosclerosis. J Inflamm Res. 2021 Dec 21;14:7143-72. [PubMed] [Google Scholar]
12. Tanase DM, Valasciuc E, Gosav EM, Ouatu A, Buliga-Finis ON, Floria M, Maranduca MA, Serban IL. Portrayal of NLRP3 inflammasome in atherosclerosis: current knowledge and therapeutic targets. Int J Mol Sci. 2023 May 3;24(9):8162. [PubMed] [Google Scholar]
13. Sun Y, Ding S. NLRP3 inflammasome in diabetic cardiomyopathy and exercise intervention. Int J Mol Sci. 2021 Dec 8;22(24):13228. [PubMed] [Google Scholar]
14. Cho S, Ying F, Sweeney G. Sterile inflammation and the NLRP3 inflammasome in cardiometabolic disease. Biomed J. 2023 Oct;46(5):100624. [PubMed] [Google Scholar]
15. Takahashi M. NLRP3 inflammasome as a key driver of vascular disease. Cardiovasc Res. 2022;118(2):372-85. [PubMed] [Google Scholar]
16. Shahi A, Afzali S, Firoozi Z, Mohaghegh P, Moravej A, Hosseinipour A, Bahmanyar M, Mansoori Y. Potential roles of NLRP3 inflammasome in the pathogenesis of Kawasaki disease. J Cell Physiol. 2023 Mar;238(3):513-32. [PubMed] [Google Scholar]

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