MCC950

MCC950 is a small molecule compound that has been investigated for its potential therapeutic effects in various inflammatory and autoimmune conditions. Its primary mechanism of action is linked to its ability to inhibit the NLRP3 inflammasome, a multiprotein complex involved in the activation of inflammatory responses.

Key points

Inhibition of NLRP3 Inflammasome:

MCC950 is known for its role as a selective inhibitor of the NLRP3 inflammasome. The NLRP3 inflammasome is implicated in several inflammatory disorders, and its dysregulation can contribute to excessive inflammation.

Anti-Inflammatory Effects:

By inhibiting the NLRP3 inflammasome, MCC950 has demonstrated anti-inflammatory effects in preclinical studies. It may modulate the release of pro-inflammatory cytokines, such as interleukin-1 beta (IL-1β), and contribute to reducing inflammation associated with various diseases.

Disease Applications:

MCC950 has been studied in the context of several inflammatory and autoimmune diseases. Research has explored its potential therapeutic applications in conditions such as rheumatoid arthritis, neuroinflammatory disorders, atherosclerosis, and other diseases where inflammasome activation plays a role.

Research and Development:

While MCC950 has shown promise in preclinical studies, its clinical development and application in humans are areas of ongoing research. Researchers are investigating its safety, efficacy, and potential side effects in various disease models.

NLRP3 Inflammasome and Diseases:

Dysregulation of the NLRP3 inflammasome has been implicated in several diseases, including autoinflammatory disorders, metabolic disorders, neurodegenerative diseases, and inflammatory bowel diseases. Inhibitors like MCC950 are being explored as potential therapeutic agents to modulate these conditions.

It's important to note that the field of inflammasome research, including the study of inhibitors like MCC950, is dynamic, and advancements continue to be made. The potential therapeutic applications of MCC950 and similar compounds are part of ongoing efforts to develop targeted treatments for inflammatory and autoimmune conditions.

The mechanism of NLRP3 inflammasome activation after subarachnoid hemorrhage is still unclear. A

study showed that TRPV1 was significantly upregulated after subarachnoid hemorrhage and was predominantly expressed in microglia/macrophages. Antagonism of TRPV1 was effective in ameliorating neurological impairment, brain edema, and neuronal damage, and reducing the inflammatory response (evidenced by reducing the number of CD16/32 positive microglia/macrophages, inhibiting the expression of CD16, CD32, CD86, IL-1b, TNF-a and blocking NLRP3 inflammasome activation). However, this effect can be abolished by NLRP3 inflammasome antagonist MCC950. In vitro experiments confirmed that TRPV1 activated NLRP3 inflammasome by increasing intracellular calcium levels. In conclusion, TRPV1 mediates EBI after SAH via calcium/NLRP3, and TRPV1 is a potential therapeutic target after subarachnoid hemorrhage [1].

1)
Zhang K, Qin Z, Chen J, Guo G, Jiang X, Wang F, Zhuang J, Zhang Z. TRPV1 modulated NLRP3 inflammasome activation via calcium in experimental subarachnoid hemorrhage. Aging (Albany NY). 2024 Jan 4;15. doi: 10.18632/aging.205379. Epub ahead of print. PMID: 38180747.