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**CRYAB inhibits inflammatory response in rat heart cell and alleviates LPS-induced injury via NF-κB/MAPK pathway in vivo and in vitro**

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**Abstract:**

**Background/Objective:** Inflammation leads to cardiovascular disease and increases the risk of heart failure. As a member of the small heat shock protein family, alpha B-crystallin (CRYAB) has been shown to be protective in inflammation response. However, the relationship between CRYAB and heart inflammation remains unclear. This study was to investigate the ability of CRYAB to alleviate LPS-induced injury in rat heart cell *in vivo* and *in vitro*.

**Methods:**High-throughput sequencing was primarily conducted to evaluate the transcriptomic changes between normal and CRYAB-overexpressing H9C2 cells after 12 h of LPS treatment. Changes in the NF-κB and MAPK pathway and cytokines were detected in both cells treated with LPS (10 μg/mL) for 3, 6, or 12 h. The same parameters were evaluated in CRYAB-knockdown H9C2 cells and CRYAB-knockout mice treated with LPS for the same period.

**Results:** Forced expression of CRYAB suppressed proinflammatory cytokines (e.g., TNF-α, IL-6 and MCP-1) expression by inhibiting the activation of NF-κB and MAPK pathway post-LPS challenge. Moreover, the lack of CRYAB expression remarkably enhanced the inflammatory responses. CRYAB knockout mice exhibited more severe pathological changes during LPS challenge.

**Conclusion:** These results indicate that CRYAB may have the ability to reduce LPS-induced inflammation in the heart and thereby represent a potential strategy for the treatment of inflammation-induced cardiac dysfunction.

**Keywords:** alpha B-crystallin; LPS; inflammation; knockout mice