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# Letter to the Editor Regarding: "Potential Protective Effects of Boldine in Rat with an Experimental Myocardial Ischemia-Reperfusion Model"

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**Keywords:** Cardiovascular medicine, heart, medicine

**Dear Editor,**

We read with great interest the article demonstrating the cardioprotective effects of boldine in a rat model of myocardial ischemia–reperfusion[1]. The authors reported that boldine administration at the onset of reperfusion improved oxidative stress indices (total antioxidant status, total oxidant status, oxidative stress index) and attenuated histopathological damage, providing valuable experimental evidence of boldine's protective potential in acute reperfusion injury.

From a technical perspective, the applied protocol (30 minutes of left anterior descending artery ligation followed by 120 minutes of reperfusion) is a well-established method to assess acute injury. A single intraperitoneal dose allowed the evaluation of early effects; however, future studies incorporating longer reperfusion periods, dose-response designs, and additional biochemical markers—such as superoxide dismutase, catalase, glutathione peroxidase, malondialdehyde, and apoptotic or inflammatory mediators including nuclear factor kappa B, caspase-3, and BAX—would provide greater mechanistic depth.

Recent preclinical data further support and complement these findings, showing that boldine reduces lipid peroxidation, enhances antioxidant enzyme activity, mitigates ventricular fibrosis, and improves functional parameters in adrenergic overload models[2].

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Moreover, emerging pharmacological insights suggest that its effects may extend beyond conventional oxidative and inflammatory pathways. In particular, connexin hemichannel blockade, suppression of inflammasome activation (interleukin-1 beta caspase-1), and improved nitric oxide bioavailability have been proposed as key mechanisms that could account for the structural and microvascular benefits observed in experimental settings[3].

In conclusion, the current body of evidence indicates that boldine exerts cardioprotective actions through multiple complementary mechanisms, ranging from antioxidant and anti-inflammatory activity to modulation of fibrosis, microvascular function, and intercellular signaling. Future studies incorporating longer reperfusion protocols, dose–response analyses, and expanded molecular endpoints will be essential to further clarify its translational potential in cardiovascular pharmacology.

Sincerely,

**Footnotes**

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## References

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