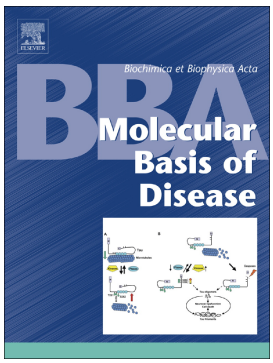

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Monoamine oxidase-dependent histamine catabolism accounts for post-ischemic cardiac redox imbalance and injury

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Keywords: monoamine oxidase; oxidative stress; histamine catabolism; cardiac post-ischemic reperfusion; mitochondria.

Highlights

- N^1-methylhistamine fuels MAO-dependent ROS production in cardiac injury
- MAO inhibition causes N^1-methylhistamine accumulation and oxidative stress decrease
- Synaptic terminals innervating the heart are relevant sources of N^1-methylhistamine
- In isolated cardiomyocytes histamine promotes ROS formation in a MAO-dependent manner

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