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ERRATUM

Hydrogen sulfide inhibits myocardial injury induced by homocysteine in rats

Lin Chang · Bin Geng · Fang Yu · Jing Zhao · Hongfeng Jiang · Junbao Du · Chaoshu Tang

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The present Fig. 6b shows that H_2S was added at early time points (i.e.100 time/S). This was an oversight. Hcy induced a transient superoxide anion release in isolated myocardial mitochondria. However, in the early times, we found that H_2S could not be added and a curve was not observed. So, we pre-incubated mitochondria with different concentrations of H_2S, followed by the addition of Hcy. We found that pre-treatment with H_2S at 10^{-9} mol/L, mostly blocked superoxide anion release induced by Hcy, and treated with H_2S from 10^{-8} to 10^{-4} mol/L completely abolished superoxide anion production, that is, the tracings were not elevated and remained at baseline.

The correct Fig. 6 is given below:

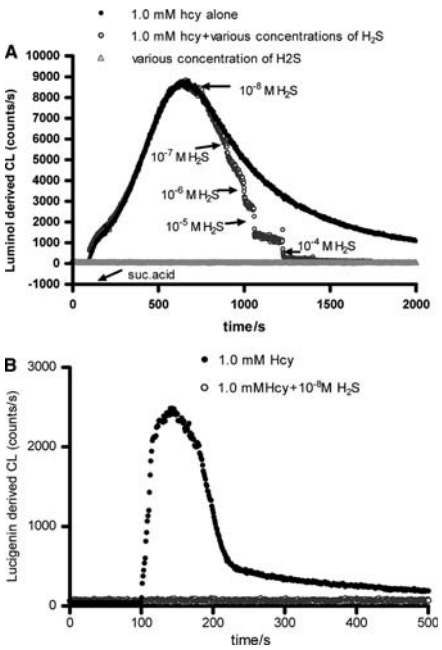

Fig. 6 Hydrogen sulfide cleaved reactive oxygen species produced by Hcy in myocardial mitochondria. **a** Myocardial mitochondria were isolated from normal rat hearts and incubated with 0.1 mmol/L Hcy for 5 min followed by H_2O_2 production triggered by succinate acid. At peak H_2O_2 production, various dosages of H_2S were added in the incubation buffer, step by step, from low to high dosage. The H_2O_2 production curve was monitored by computerized chemilal machine. **b** Isolated myocardial mitochondria were incubated with 0.1 mmol/L Hcy for 5 min followed by measuring the superoxide anion production by lucigenin-derived chemiluminescence. At the peak of the curve, different concentrations of H_2S were added and the alteration in superoxide anion was recorded. An amount of 0.1 mM hypoxanthine plus 10 mU xanthine oxidase induced superoxide anion was used as a positive control

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L. Chang · B. Geng (✉) · C. Tang
Institute of Cardiovascular Research, Peking University,
Xishuku Street 8, West District, 100034 Beijing,
People's Republic of China
e-mail: bingeng@bjmu.edu.cn

B. Geng · F. Yu · J. Zhao · H. Jiang · C. Tang
Department of Physiology,
Peking University Health Science Center,
Beijing, People's Republic of China

J. Du
Department of Pediatric, First Hospital of Peking University,
Beijing, People's Republic of China