

## Hydrogen sulfide inhibits myocardial injury induced by homocysteine in rats

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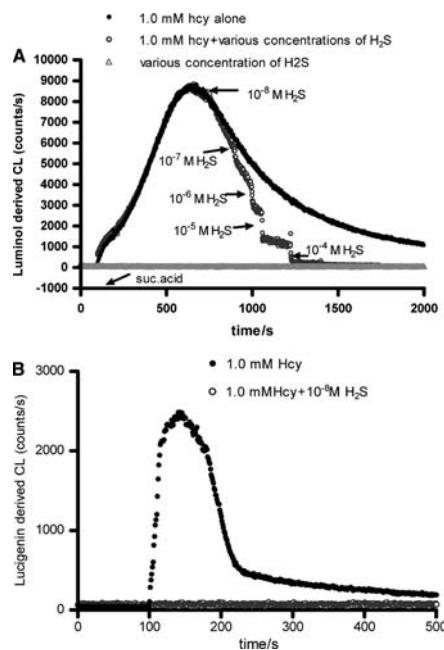
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### Erratum to: Amino Acids

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The present Fig. 6b shows that H<sub>2</sub>S 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<sub>2</sub>S could not be added and a curve was not observed. So, we pre-incubated mitochondria with different concentrations of H<sub>2</sub>S, followed by the addition of Hcy. We found that pre-treatment with H<sub>2</sub>S at 10<sup>-9</sup> mol/L, mostly blocked superoxide anion release induced by Hcy, and treated with H<sub>2</sub>S from 10<sup>-8</sup> to 10<sup>-4</sup> 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<sub>2</sub>O<sub>2</sub> production triggered by succinate acid. At peak H<sub>2</sub>O<sub>2</sub> production, various dosages of H<sub>2</sub>S were added in the incubation buffer, step by step, from low to high dosage. The H<sub>2</sub>O<sub>2</sub> production curve was monitored by computerized chemiluminescence 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<sub>2</sub>S 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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