

ERRATUM: Regulation of heart function by endogenous gaseous mediators-crosstalk between nitric oxide and hydrogen sulfide by Yong QC, Cheong JL, Hua F, Deng LW, Khoo YM, Lee HS, Perry A, Wood M, Whiteman M, Bian JS. *Antioxid Redox Signal* 14:2081-2091, 2011.

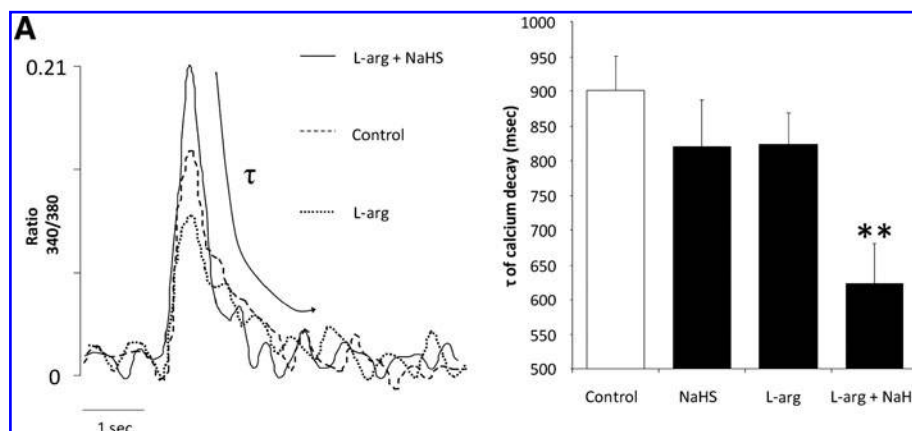
In the June 2011 issue of *Antioxidants & Redox Signaling* (vol. 14, no. 11; 2081–2091), there is an error in Figure 3A, in the article “Regulation of heart function by endogenous gaseous mediators-crosstalk between nitric oxide and hydrogen sulfide.”

The authors noticed the inaccuracy after recently checking past experimental data.

The correct version of Figure 3A is provided.

The experiment was done together with several nitric oxide (NO) donors, including L-arginine and sodium nitroprusside (SNP). The mistake occurred during manuscript preparation, when the experimental data using SNP as NO donor were erroneously plotted as L-arginine. In this correction, we replaced figure 3A with the correct L-arginine data. The representative tracings were also edited to reflect the L-arginine data. The authors would like to stress that only the p-value of L-arg + NaHS group remains significant ($p < 0.05$) when compared against the control. Therefore, the authors claim that these changes do not alter the conclusions of this article.

Incorrect Fig. 3A



Corrected Fig. 3A

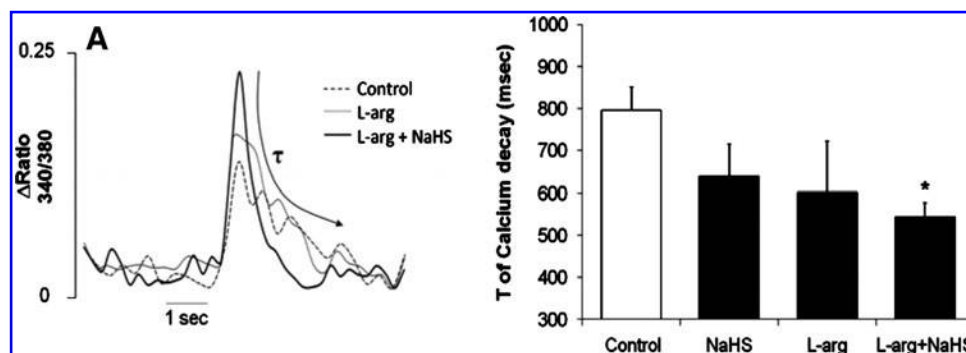


FIG. 3A Representative tracings and group data showing the effect of L-arg and L-arg+NaHS on the decay time (t) of $EL-[Ca^{2+}]_i$ transients in rat ventricular myocytes. L-arg+NaHS significantly shortened the decay time, indicating that L-arg+NaHS increased the velocity of calcium decay. Mean \pm SEM; $n=5-9$; * $p < 0.05$, versus control group.

The authors regret this error and apologize for any confusion or inconvenience it may have caused.

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