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Erratum: Sarcoendoplasmic Reticulum Ca²⁺ ATPase. A Critical Target In Chlorine Inhalation-induced Cardiotoxicity

The authors of Ahmad and colleagues (1), published in the April 2015 issue of *AJRCMB*, wish to correct an error in their article. In Figure 4, a duplicate of panel B2 was inadvertently included instead of panel B6. A revised version of Figure 4 is included here that includes the correct version of panel B6.

The authors apologize to the *Journal's* readers for any confusion caused by this error.

Reference

 Ahmad S, Ahmad A, Hendry-Hofer TB, Loader JE, Claycomb WC, Mozziconacci O, Schöneich C, Reisdorph N, Powell RL, Chandler JD, Day BJ, Veress LA, White CW. Sarcoendoplasmic reticulum Ca²⁺ ATPase. A critical target in chlorine inhalation-induced cardiotoxicity. *Am J Respir Cell Mol Biol* 2015;52:492–502.

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9

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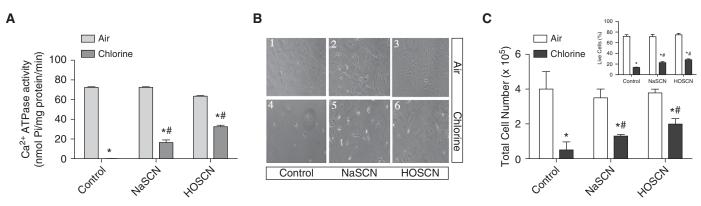


Figure 4 [revised]. Effects of sodium thiocyanate (NaSCN) and hypothiocyanate (HOSCN) on SERCA inactivation and cell death due to chlorine. Cardiomyocytes were isolated and cultured from naive rat hearts, as described in MATERIALS AND METHODS. Approximately 50×10^4 cells/well were plated on laminin-coated 12-well plates and then exposed to 0 or 50 ppm chlorine in an *in vitro* exposure chamber, as described in MATERIALS AND METHODS. Cell were treated with NaSCN (500 μ M) or HOSCN (100 μ M; freshly prepared) 10 minutes before chlorine exposures, as described in MATERIALS AND METHODS. (*A*) Ca²⁺ATPase activity was determined in whole-cell homogenates prepared in SERCA activity buffer (detailed in MATERIALS AND METHODS). Values shown are mean \pm SEM (*n* = 4). *Significant difference (*P* < 0.05) from control (0 ppm chlorine); [#]significant difference from chlorine-exposed cells without pretreatment. (*B*) A representative pictogram illustrating cardiomyocyte morphology in these experiments. (*C*) Total cell counts (live cells only) obtained in cardiomyocyte cultures after exposure to 0 ppm or 50 ppm chlorine, both in the presence and absence of NaSCN and HOSCN. The *inset* in (*C*) shows SCN-induced rescue of chlorine-exposed cardiomyocytes where NaSCN or HOSCN were supplemented 10 minutes after chlorine exposure. Percent live cells that excluded trypan blue were counted in a Bio-Rad cell counter. Values shown are mean \pm SEM (*n* = 4). *Significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference (*P* < 0.05) from control (0 ppm Cl₂); [#]significant difference from chlorine-exposed cells without pretreatment.

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Erratum: PD-1/PD-L1 Pathway Mediates the Alleviation of Pulmonary Fibrosis by Human Mesenchymal Stem Cells in Humanized Mice

The authors wish to correct an error in Figure 3D in their article published in the June 2018 issue of the *Journal* (1).

The authors were informed of the presence of identical regions in the images of α -SMA (α -smooth muscle actin) staining in the BLM (bleomycin) column in Figure 3D, and α -SMA in the Humanized+BLM column in Figure 1D. Both images correspond to the same treatment group at the same time point, so similarities are to be expected. However, it was not the intention of the authors to use the same image in both figures. The overlap was the result of accidentally picking two representative images from the same slide for α -SMA staining; these images contained partial overlap. In the revised version of Figure 3D, the original image of α -SMA staining has been replaced by a new representative picture that lacks overlap. This correction does not affect the results or the conclusions of this work, as both images were from the same experimental group.

The authors apologize to the *Journal* and its readers for any confusion caused by this error. ■

Reference

 Ni K, Liu M, Zheng J, Wen L, Chen Q, Xiang Z, Lam KT, Liu Y, Chan GCF, Lau YL, Tu W. PD-1/PD-L1 pathway mediates the alleviation of pulmonary fibrosis by human mesenchymal stem cells in humanized mice. *Am J Respir Cell Mol Biol* 2018;58: 684–695.

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