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Erratum to “Evidence for a receptor-activated Ca^{2+} entry pathway independent from Ca^{2+} store depletion in endothelial cells” [*Cell Calcium* 43 (1) (2008) 83—94]

H. Jousset^a, R. Malli^b, N. Girardin^a, W.F. Graier^b, N. Demaurex^a, M. Frieden^{a,*}

^aDepartment of Cell Physiology and Metabolism, Geneva Medical Center, 1 rue Michel Servet, 1211 Geneva 4, Switzerland ^bInstitute of Molecular Biology and Biochemistry, Center of Molecular Medicine, Medical University Graz, 8010 Graz, Austria

In our previous paper [1], we reported that La^{3+} was a useful tool to discriminate between RACE (receptor-activated Ca^{2+} entry) and SOCE (store-operated Ca^{2+} entry) in the human umbilical vein endothelial cell line, EA.hy926. In particular at 10 μM , La^{3+} almost fully blocked SOCE (activated by thapsigargin, TG) while it appeared to have no effect on RACE (activated by histamine). In course of the continuation of this work, our experiments revealed that the lack of inhibitory effect of La^{3+} on RACE was a regrettable artifact, due to the presence of phosphate in the histamine batch we were using. Indeed the batch of histamine was a phosphate salt and not a chloride salt. Testing the block of La^{3+} on the histamine—Cl response revealed that La^{3+} is equally efficient to inhibit the RACE than reported on SOCE. This suggests that the phosphate formed a complex with La^{3+} , thus precluding its inhibitory effect on histamine— PO_4 induced Ca^{2+} entry. However, the other evidence presented in the paper in favor of two separate Ca^{2+} entry pathways in endothelial cells were confirmed and are still valid.

References

- [1]. Jousset H, Malli R, Girardin N, Graier WF, Demaurex N, Frieden M. Evidence for a receptor-activated Ca^{2+} entry pathway independent from Ca^{2+} store depletion in endothelial cells. *Cell Calcium*. 2008; 43:83–94. [PubMed: 17548108]

*Corresponding author at: Department of Cell Physiology and Metabolism, University of Geneva Medical School, 1 Michel-Servet, 1211 Geneva 4, Switzerland. Tel.: +41 22 379 5198; fax: +41 22 379 5338. maud.frieden@unige.ch (M. Frieden).

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