Corrigendum

Substance P stimulates human airway submucosal gland secretion mainly via a CFTR-dependent process

Jae Young Choi, Monal Khansaheb, Nam Soo Joo, Mauri E. Krouse, Robert C. Robbins, David Weill, and Jeffrey J. Wine


Citation for this corrigendum: J Clin Invest. 2010;120(3):931–932. doi:10.1172/JCI37284C1.

Following the publication of this manuscript, the authors discovered carbachol contamination of an aliquot of substance P used to generate the data in Figures 7A and 7B in the published version of this work. The authors have performed the relevant experiments again with a fresh, uncontaminated aliquot of substance P. The previously published data and the corrected data are compared in Table 3 below. The corrected text describing the new data for the Results section and the corrected Figure 7 appear below. The authors confirm that the conclusions of their study remain unchanged.

In unstimulated cells, $[\text{Ca}^{2+}]_i$ was 70–140 nM. SubP increased $[\text{Ca}^{2+}]_i$ in 47 of 58 cells from 8 subjects by 133 ± 35 nM (peak value). All 58 cells responded to carbachol with increases in $[\text{Ca}^{2+}]_i$ that were larger than those to SubP, the responses to 1 and 10 μM carbachol were 186 ± 17 nM and 231 ± 36 nM, respectively. We considered the possibility that gland cells that are unresponsive to SubP might be a different cell type. To help differentiate serous and mucous cells in some of the dispersed cell preparations, we used PAS staining and observed a negative correlation between PAS reactivity and SubP responsiveness. For SubP-responsive cells, 7 of 25 (28%) were PAS positive (contain mucus), while for SubP-nonresponsive cells, 6 of 8 (75%) were PAS positive.

The authors regret the errors.

Figure 7
Evidence that SubP stimulates gland secretion, in part, via elevating $[\text{Ca}^{2+}]_i$. (A) Fluorescence changes in response to 10 μM SubP and 10 μM carbachol. Cell diameters in images are approximately 20 microns. (B) $[\text{Ca}^{2+}]_i$ versus time for 10 cells from images in A, measured in response to sequential pulses of 10 μM SubP and 10 μM carbachol. (C) $[\text{Ca}^{2+}]_i$ versus time for 10 cells from images in A, measured in response to sequential pulses of 10 μM SubP without and with 5 μM atropine (Atr). Fluorescence ratio, 340 nm/380 nm. (D) Mean response to SubP in presence or absence of BAPTA-AM (500 μM); 4 experiments from 2 HN and 1 DC subjects (16–20 glands). Error bars are SEM. (E) Mean response to SubP in the absence and presence of clotrimazole (25 μM), which blocks Ca2+-activated K+ channels ($n = 4$, 27–42 glands). *P < 0.05 versus SubP responses. Error bars are SEM.
Table 3
Summary of original and new experiments with Substance P and intracellular calcium

<table>
<thead>
<tr>
<th></th>
<th>I Original experiment with suspect SubP</th>
<th>II Original experiments with good SubP</th>
<th>III New experiments</th>
<th>I + II Published data</th>
<th>II + III Corrected data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting [Ca^{2+}]_i</td>
<td>72–105 nM</td>
<td>70–120nM</td>
<td>85–140 nM</td>
<td>70–120nM</td>
<td>70–140 nM</td>
</tr>
<tr>
<td>n, subjects; n cells</td>
<td>1; 6</td>
<td>4; 33</td>
<td>4; 25</td>
<td>5; 39</td>
<td>8; 58</td>
</tr>
<tr>
<td>n (%) cells responding to SubP</td>
<td>6/6 (100%)^A</td>
<td>25/33 (76%)</td>
<td>22/25 (88%)</td>
<td>31/39 (79%)</td>
<td>47/58 (81%)</td>
</tr>
<tr>
<td>[Ca^{2+}]_i response to SubP (peak value, nM)</td>
<td>134 ± 34</td>
<td>140 ± 32</td>
<td>125 ± 18</td>
<td>139 ± 33</td>
<td>133 ± 35</td>
</tr>
<tr>
<td>n cells responding to carbachol (1 or 10 μM)</td>
<td>6/6 (100%)</td>
<td>33/33 (100%)</td>
<td>25/25 (100%)</td>
<td>39/39 (100%)</td>
<td>58/58 (100%)</td>
</tr>
<tr>
<td>[Ca^{2+}]_i response to Carb 1 μM (peak value, nM)</td>
<td>194 ± 10</td>
<td>186 ± 17</td>
<td>Not done</td>
<td>187± 19</td>
<td>186± 17</td>
</tr>
<tr>
<td>[Ca^{2+}]_i response to Carb 10 μM (peak value, nM)</td>
<td>256 ± 20</td>
<td>252 ± 15</td>
<td>202 ± 35</td>
<td>253 ± 17</td>
<td>231 ± 36</td>
</tr>
<tr>
<td>PAS positivity in SubP responsive cells</td>
<td>2/6 (33%)</td>
<td>7/25 (28%)</td>
<td>Not done</td>
<td>9/31 (29%)</td>
<td>7/25 (28%)</td>
</tr>
<tr>
<td>PAS positivity in SubP nonresponsive cells</td>
<td>Not done</td>
<td>6/8 (75%)</td>
<td>Not done</td>
<td>6/8 (75%)</td>
<td>6/8 (75%)</td>
</tr>
<tr>
<td>% cells responding to SubP (10 μM) in presence of atropine</td>
<td>Not done</td>
<td>Not done</td>
<td>22/22 (100%)</td>
<td>Not done</td>
<td>22/22 (100%)</td>
</tr>
</tbody>
</table>

^APublished data generated using possibly contaminated Substance P.

Corrigendum

FoxO1 expression in osteoblasts regulates glucose homeostasis through regulation of osteocalcin in mice


Citation for this corrigendum: J Clin Invest. 2010;120(3):932. doi:10.1172/JCI39901C1.

The legend for Figure 5H was incorrect. The correct text appears below.

(H) Changes in uncarboxylated or undercarboxylated Ocn in serum of WT and Foxo1+/– mice; n = 5 mice/group.

The authors regret the error.

Erratum

Urea-induced ROS generation causes insulin resistance in mice with chronic renal failure

Maria D’Apolito, Xueliang Du, Haihong Zong, Alessandra Catucci, Luigi Maiuri, Tiziana Trivisano, Massimo Pettoello-Mantovani, Angelo Campanozzi, Valeria Raia, Jeffrey E. Pessin, Michael Brownlee, and Ida Giardino


Citation for this erratum: J Clin Invest. 2010;120(3):932. doi:10.1172/JCI37672E1.

During the preparation of the manuscript, the urea infusion rate was incorrectly given. The correct sentence containing the infusion rate appears below.

During the preparation of the manuscript, the urea infusion rate was incorrectly given. The correct sentence containing the infusion rate appears below.

The rats were allowed to recover from the surgery for 5 days and then were either infused with isotonic PBS or urea (100 mg/kg/h) for 48 hours using a microdialysis pump.

The JCI regrets the error.