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Corrigendum

Inhibitory effects of calcitonin gene-related peptide on substance-P-induced superoxide production in human neutrophils [Eur. J. Pharmacol. 314 (1996) 175–183] [1]

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In the above-mentioned paper, Figs. 3 and 4 were interchanged, and panels E and F of Fig. 6 were omitted. On the following pages, please find therefore a complete set of the correct Figs. 1–6.

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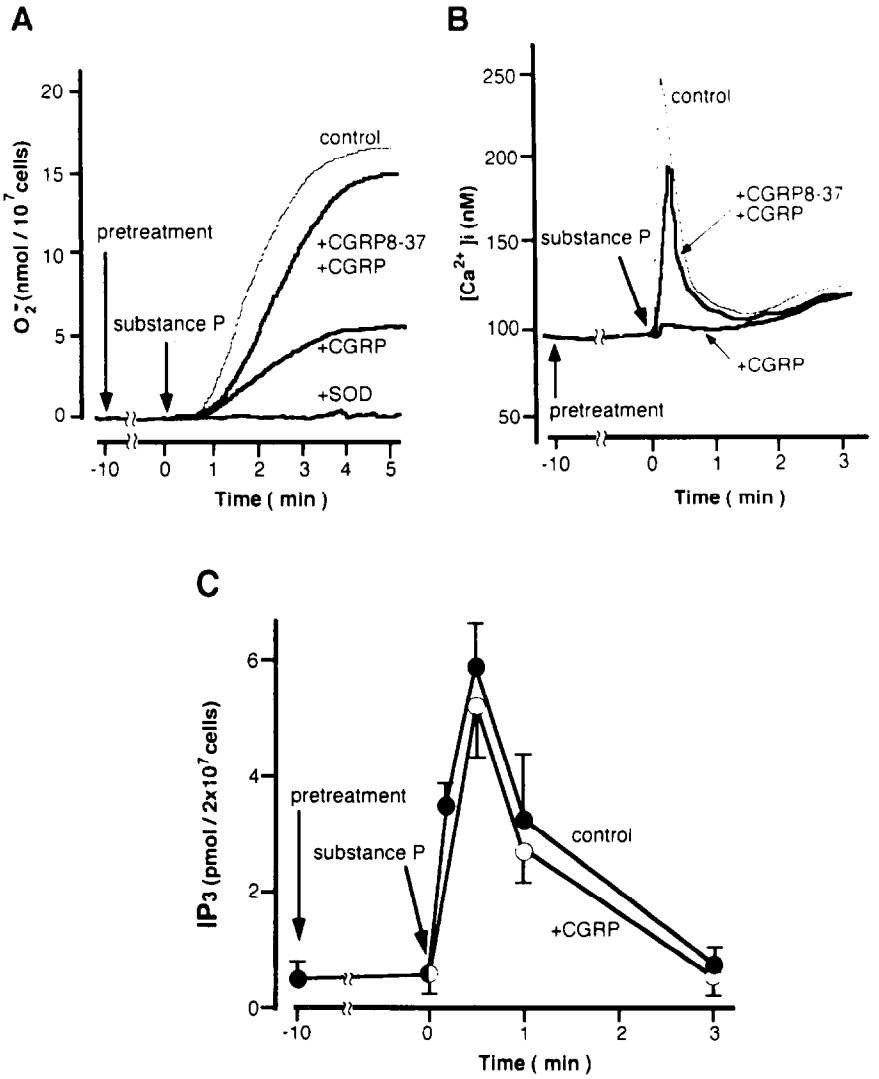

Fig. 1. Effects of CGRP on substance-P-induced O_2^- production (A), changes in $[Ca^{2+}]_i$ (B) and IP_3 formation (C) in human neutrophils. (A) A representative time-course of O_2^- production. O_2^- production was monitored spectrophotometrically as described in Materials and methods. Cells were equilibrated for 10 min in HBSS medium with or without (control) test reagents; 10 μM CGRP, 10 μM CGRP plus a CGRP receptor antagonist, 10 μM CGRP-(8–37), or 400 units/ml of superoxide dismutase (SOD), and then exposed to 30 μM substance P. (B) A representative trace of $[Ca^{2+}]_i$. Cells loaded with fura-2-AM were equilibrated for 10 min in HBSS medium with or without (control) test reagents; 10 μM CGRP or 10 μM CGRP plus 10 μM CGRP-(8–37), and then exposed to 30 μM substance P. (C) A representative time-course of IP_3 formation. IP_3 formation was measured by using an IP_3 radioreceptor-assay kit as described in Materials and methods. Cells were equilibrated for 10 min in HBSS medium with or without (control) test reagents; 10 μM CGRP, and then exposed to 30 μM substance P.

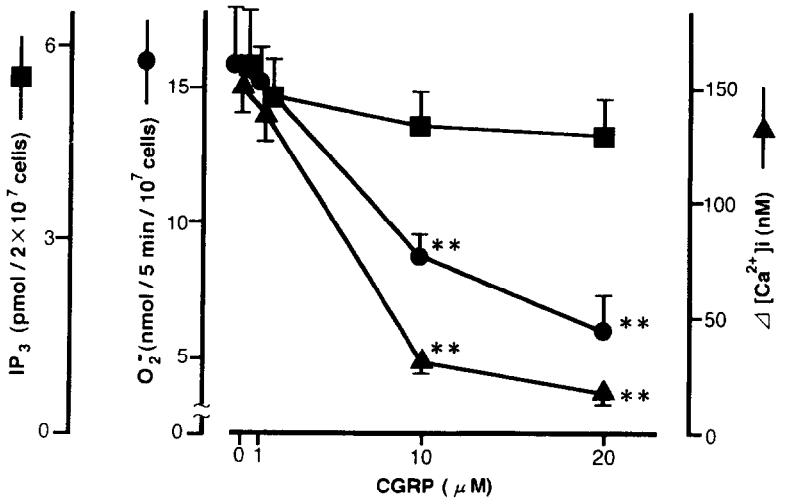

Fig. 2. Dose-dependent effects of CGRP on O_2^- production, $\Delta[Ca^{2+}]_i$ and IP_3 formation in neutrophils stimulated with substance P. Human neutrophils were preincubated for 10 min with HBSS medium with or without 1, 10 or 20 μM CGRP, and then exposed to 30 μM substance P. Each symbol represents the mean ± S.E. for 16–20 preparations. ** $P < 0.01$, compared to the value without CGRP.

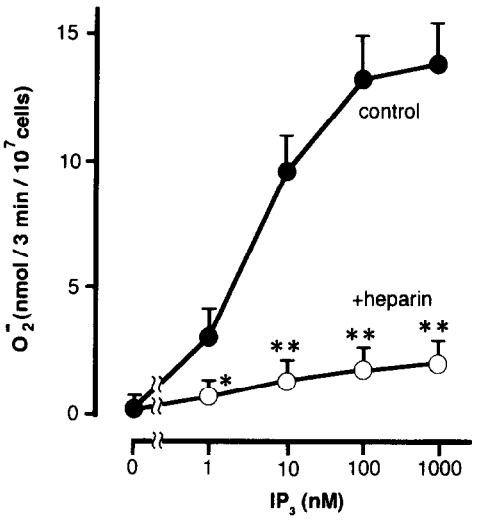

Fig. 3. The concentration-response curve for O_2^- production in permeabilized neutrophils stimulated with IP_3. The saponin-permeabilized human neutrophils were preincubated for 10 min with (○) or without (●) an IP_3 receptor antagonist, heparin (2 μg/ml), and then exposed to various concentrations of IP_3. The production of O_2^- was calculated from the amounts of cytochrome *c* reduced 3 min after the addition of IP_3. Each symbol with a bar represents the mean ± S.E. for 10–12 preparations.

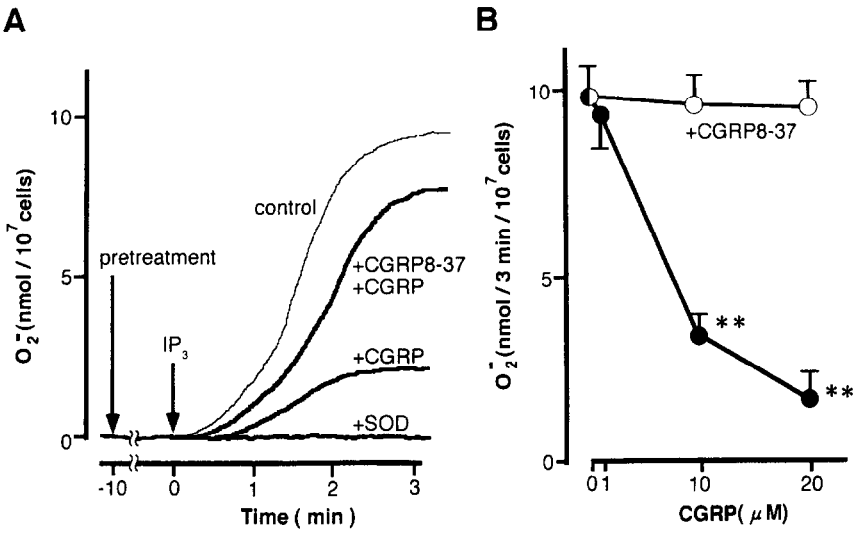

Fig. 4. Effects of CGRP on IP_3-induced O_2^- production in permeabilized human neutrophils. (A) A representative time-course of O_2^- production. O_2^- production was monitored spectrophotometrically as described in Materials and methods. Saponin-permeabilized cells were preincubated for 10 min with or without (control) 10 μM CGRP, 10 μM CGRP plus a CGRP receptor antagonist, 10 μM CGRP-(8–37) or 400 units/ml of superoxide dismutase (SOD), and then exposed to 10 nM IP_3. (B) Dose-dependent effects of CGRP on IP_3-induced O_2^- production. The permeabilized cells were preincubated for 10 min with or without different concentrations of CGRP (●) or CGRP plus 10 μM CGRP-(8–37) (○), and then exposed to 10 nM IP_3. Each symbol with a bar represents the mean ± S.E. for 16–20 preparations. $^{**}P < 0.01$ compared with the value of without CGRP.

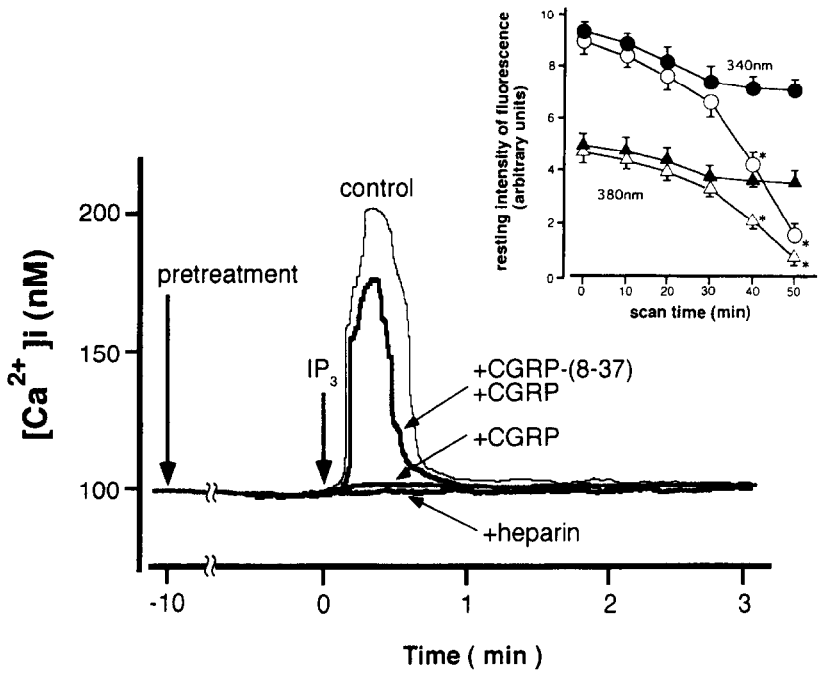

Fig. 5. Representative traces of IP_3-induced changes in $[Ca^{2+}]_i$ in permeabilized human neutrophils. $[Ca^{2+}]_i$ was measured fluorometrically as described in Materials and methods. Saponin-permeabilized and fura-2-AM-loaded cells were preincubated for 10 min with or without (control) 10 μM CGRP, 10 μM CGRP plus a CGRP antagonist, 10 μM CGRP-(8–37), or an IP_3 receptor antagonist, 2 μg/ml heparin, and then exposed to 10 nM IP_3. Inset: the time-course of changes in resting fura-2 fluorescence intensities at 340 nm (●, ○) and 380 nm (▲, △) of the single saponin-permeabilized (○, △) and non-permeabilized (●, ▲) cells were checked by using an ultrasensitive fluorescence counting imaging camera as described in Materials and methods. Each symbol with a bar represents the mean ± S.E. for 8–10 preparations. $^{*}P < 0.01$ compared to the value of the non-permeabilized cell.

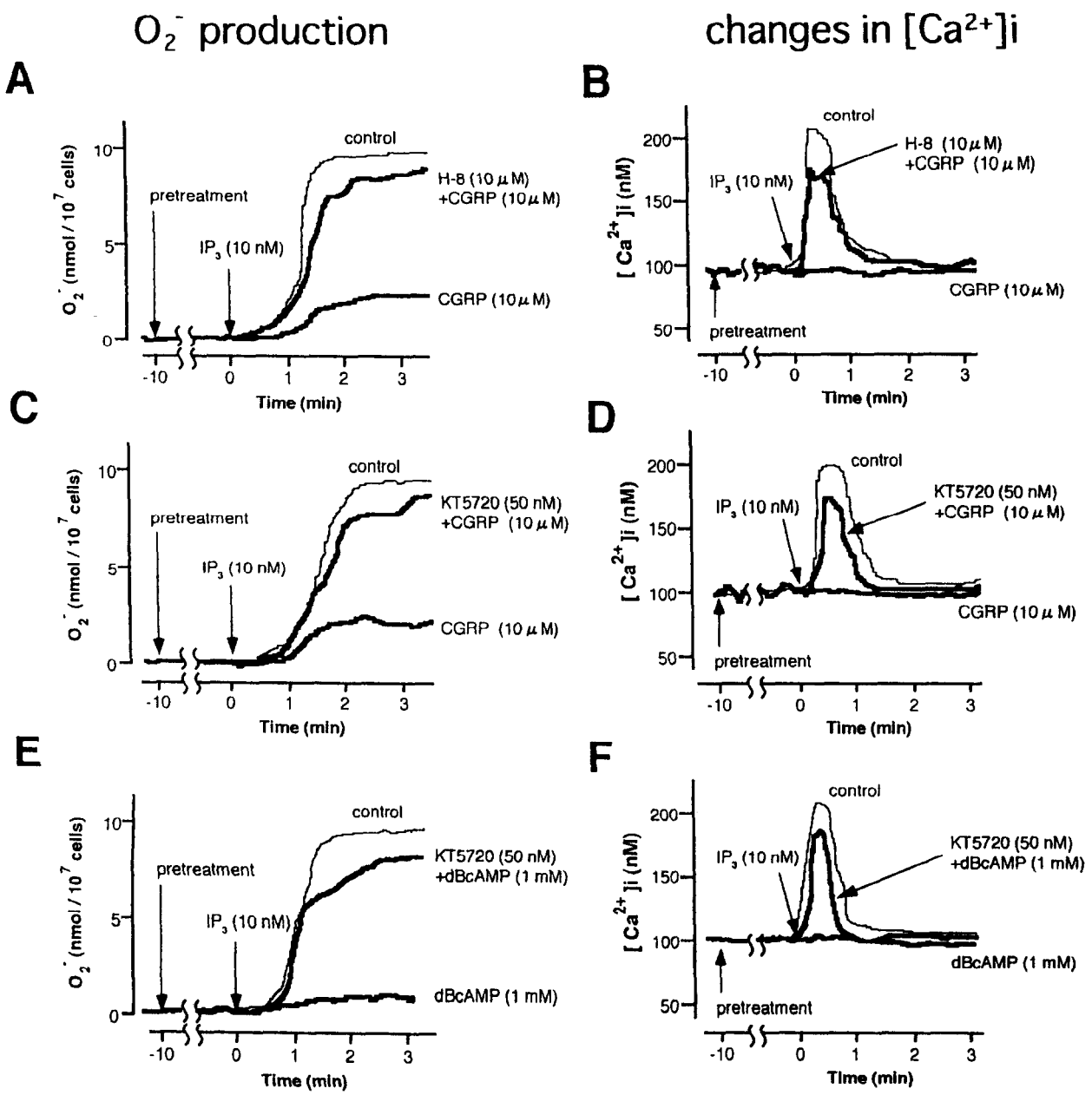

Fig. 6. Effects of cAMP-dependent protein kinase inhibitors on the inhibitory effects of CGRP and dibutyl cAMP in IP_3-induced O_2^- production and $[Ca^{2+}]_i$ changes. Saponin-permeabilized human neutrophils were pretreated for 10 min with or without (control) 10 μM CGRP or 10 μM CGRP plus either of the cAMP-dependent protein kinase inhibitors, 10 μM H-8 (A,B) or 50 nM KT5720 (C,D), and then exposed to 10 nM IP_3. Same experiments carried out by using 1 mM dibutyl cAMP (E,F). O_2^- production (A,C,E) and changes in $[Ca^{2+}]_i$ (B,D,F) were measured as described in Materials and methods.