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ERRATUM

Erratum to: Isoform- and receptor-specific channel property of canonical transient receptor potential (TRPC)1/4 channels

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The authors would like to apologize for inadvertently using Fig. 1d also for Fig. 5 h in the printed version of this paper.

The correct versions of Fig. 1d and Fig. 5 h are given below.

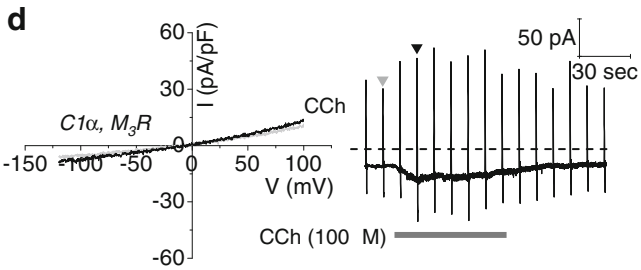

Fig. 1 $G_{q/11}$-coupled receptor stimulation and TRPC1α, TRPC1α/4, TRPC4 channel activity. From **a** to **h**, all *left panels* indicate *I–V* relationship and *right panels* indicate corresponding current traces. *Inset legends at upper left area of I–V curves* demonstrate heterologously expressed proteins. **a**, **b** In cells expressing M1 or M3 receptor, receptor stimulation by extracellular carbachol (100 μM) did not induced significant whole-cell current. **c**, **d** Cells expressing TRPC1α channels showed indistinguishable current increment in response to M1 or M3 stimulation achieved by extracellular carbachol (*CCh*, 100 μM). **e**, **f** Heteromeric TRPC1α/4 channel showed typical outward-rectifying *I–V* curves in response to M1 or M3 stimulation. **g**, **h** Genuine TRPC4 homomeric currents (without co-expression of TRPC1α) in response to M1 or M3 receptor stimulation. **i** Left panel indicates summarized current density measured above and *right panel* indicates rectification factor ($|I_{+80\ mV}/I_{-60\ mV}|$) analysis which was devised in order to show the sheer difference in *I–V* curve signature. **j** *Left panel* compares double-rectifying *I–V* curves of CCh-activated TRPC4 (*light gray*) and outward-rectifying *I–V* curves of CCh-activated TRPC1α/4 (*black*). In order to quantify the order of outward rectification, another rectification factor ρ (Rho) was devised which parameterizes two different slope conductance: $\rho \equiv |g_{+10\sim+50mV}|/|g_{-120\sim-80\ mV}|$. Two different *I–V* curves show stark difference in ρ value

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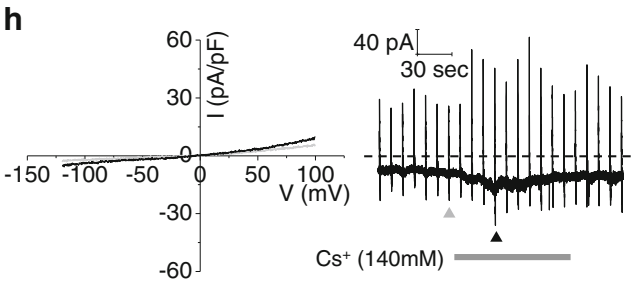

Fig. 5. TRPC1α, TRPC1α/4, and TRPC4 channel activities in response to $G_{i/o}$-coupled receptor (M2) stimulation and overexpression of $G\alpha_{i2}^{Q205L}$ protein. **a**, **b** Genuine TRPC4 homomeric channel activity in response to M2 receptor stimulation and overexpression of $G\alpha_{i2}^{Q205L}$ (intrinsic GTPase activity deficient mutant) protein. Extracellular carbachol (*CCh*, 100 μM) and Cs^+ -rich solution ($[Cs^+]_o$ = 140 mM) was treated for each activation systems, respectively. TRPC4 channels activated by each condition showed typical double-rectifying *I–V* curves. **c**, **d** TRPC1α homomeric channel activities in response to M2 receptor and $G\alpha_{i2}^{Q205L}$ protein. Neither M2 receptor stimulation nor $G\alpha_{i2}^{Q205L}$ protein activated TRPC1α channels. **e**, **f** TRPC1α/4 heteromeric channel activities in response to M2 receptor and $G\alpha_{i2}^{Q205L}$ protein. Each activation system was insufficient to induce TRPC1α/4 channel currents but selectively activated TRPC4 homomeric channels. **g**, **h** TRPC1β homomeric channel activities in response to M2 receptor and $G\alpha_{i2}^{Q205L}$ protein. Both systems could not induce channel activation. **i**, **j** TRPC1β/4 heteromeric channel activities in response toM2 receptor and $G\alpha_{i2}^{Q205L}$ protein. Like TRPC1α/4, TRPC1β/4 did not permit their activation to those stimulators but only TRPC4 did permit their activation