

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

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Published online: 3 December 2013
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Erratum to: Pflügers Arch - Eur J Physiol
DOI 10.1007/s00424-013-1332-y

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

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

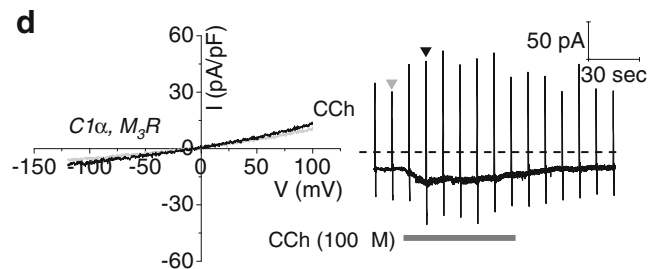


Fig. 1 $G_{q/11}$ -coupled receptor stimulation and TRPC1 α , TRPC1 $\alpha/4$, TRPC4 channel activity. From **a** to **h**, all *left panels* indicate $I-V$ relationship and *right panels* indicate corresponding current traces. *Inset legends at upper left area of $I-V$ curves* demonstrate heterologously expressed proteins. **a, b** In cells expressing M1 or M3 receptor, receptor stimulation by extracellular carbachol (CCh, 100 μ M) did not induced significant whole-cell current. **c, d** Cells expressing TRPC1 α channels showed indistinguishable current increment in response to M1 or M3 stimulation achieved by extracellular carbachol (CCh, 100 μ M). **e, f** Heteromeric TRPC1 $\alpha/4$ 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 TRPC1 α) in response to M1 or M3 receptor stimulation. **i** Left panel indicates summarized current density measured above and *right panel* indicates rectification factor ($(I_{+80\text{ mV}}/I_{-60\text{ mV}})$) 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 TRPC1 $\alpha/4$ (*black*). In order to quantify the order of outward rectification, another rectification factor ρ (Rho) was devised which parameterizes two different slope conductance: $\rho \equiv |g_{+10 \rightarrow +50\text{ mV}}/g_{-120 \rightarrow -80\text{ mV}}|$. Two different $I-V$ curves show stark difference in ρ value

The online version of the original article can be found at <http://dx.doi.org/10.1007/s00424-013-1332-y>.

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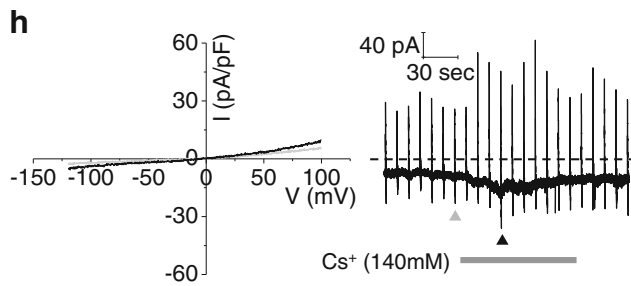


Fig. 5. TRPC1 α , TRPC1 $\alpha/4$, and TRPC4 channel activities in response to $G_{i/o}$ -coupled receptor (M2) stimulation and overexpression of $G\alpha_{12}^{Q205L}$ protein. **a, b** Genuine TRPC4 homomeric channel activity in response to M2 receptor stimulation and overexpression of $G\alpha_{12}^{Q205L}$ (intrinsic GTPase activity deficient mutant) protein. Extracellular carbachol (*CCh*, 100 μ M) 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** TRPC1 α homomeric channel activities in response to M2 receptor and $G\alpha_{12}^{Q205L}$ protein. Neither M2 receptor stimulation nor $G\alpha_{12}^{Q205L}$ protein activated TRPC1 α channels. **e, f** TRPC1 $\alpha/4$ heteromeric channel activities in response to M2 receptor and $G\alpha_{12}^{Q205L}$ protein. Each activation system was insufficient to induce TRPC1 $\alpha/4$ channel currents but selectively activated TRPC4 homomeric channels. **g, h** TRPC1 β homomeric channel activities in response to M2 receptor and $G\alpha_{12}^{Q205L}$ protein. Both systems could not induce channel activation. **i, j** TRPC1 $\beta/4$ heteromeric channel activities in response to M2 receptor and $G\alpha_{12}^{Q205L}$ protein. Like TRPC1 $\alpha/4$, TRPC1 $\beta/4$ did not permit their activation to those stimulators but only TRPC4 did permit their activation