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Cellular and Molecular Life Sciences

ERRATUM

Erratum to: Molecular and cellular mechanisms that initiate pain and itch

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Unfortunately, the original publication of this paper contained errors in the presentation of Fig. 2. The corrected Fig. 2 is given here.

The online version of the original article can be found under doi:10.1007/s00018-015-1904-4.

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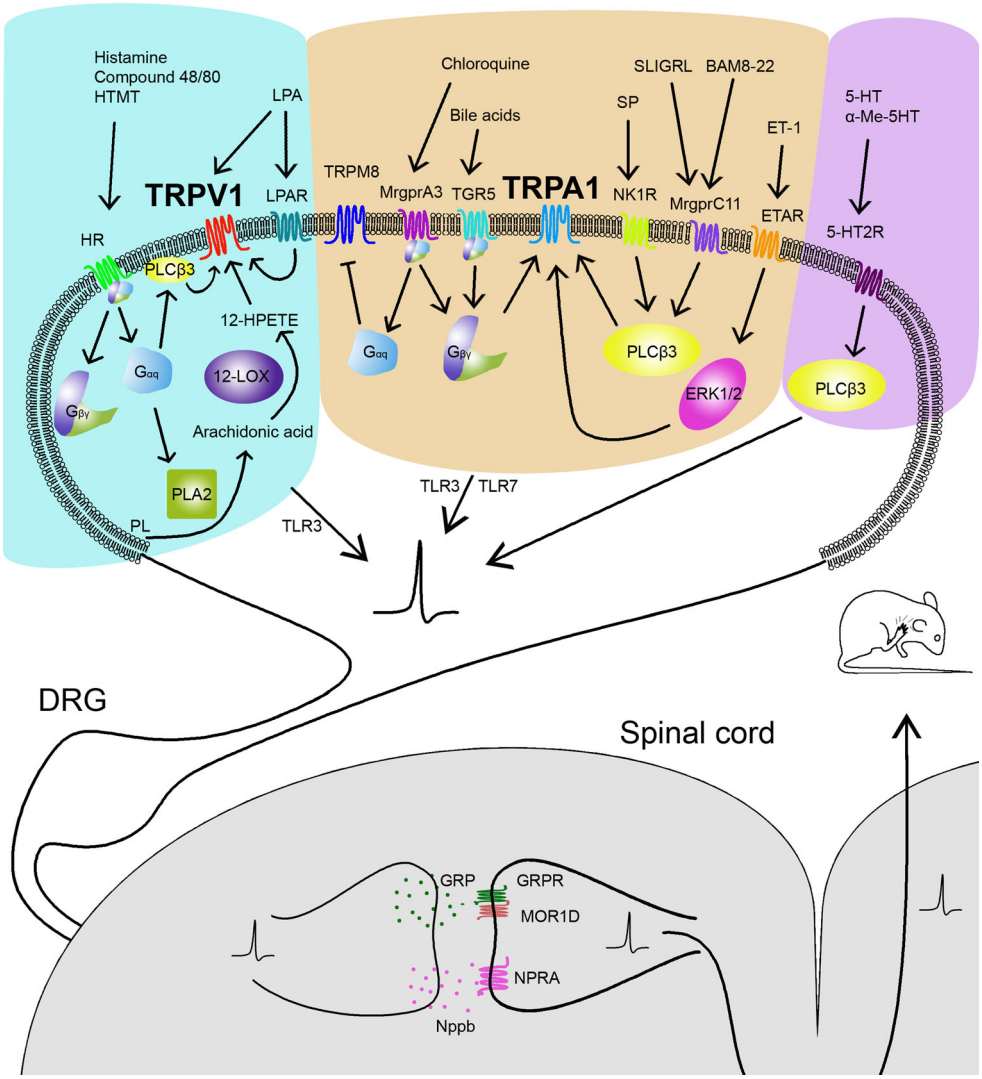

Fig. 2 Molecular basis of itch signaling. There are three classes of pruritogen-elicited itch: TRPV1-dependent, TRPA1-dependent, and non-TRPV1–TRPA1-dependent. Histamine, compound 48/80, and HTMT activate HRs, which activate TRPV1 through both PLCβ3 and PLA2/12-LOX signaling pathways. TRPV1 could also mediate LPA-induced itch, either through direct activation or indirect sensitization of TRPV1 through G protein-coupled LPAR as described for LPA-induced pain responses. TRPA1 is required for itch signaling induced by CQ, SLIGRL, BAM8-22, ET-1, SP, and bile acids, which bind to their respective receptors MrgprA3, MrgprC11, ETAR, NK1R, and TGR5. $G_{\beta\gamma}$ subunit, PLCβ3, and ERK1/2 participate in signaling downstream from these GPCRs and contribute to the activation of TRPA1. TLR3 is involved in both TRPV1- and TRPA1-dependent itch sensations, whereas TRL7 is specifically involved in TRPA1-dependent itch. The itch sensation induced by serotonin is unique since neither TRPA1 nor TRPV1 is involved, although PLCβ3 plays important roles in both 5-HTR- and TRPA1-dependent itch. In the spinal cord, presynaptically released GRP and Nppb relay itch signals to the second-order spinal neurons by binding to corresponding heteromeric GRPR/MOR1D and NPRA receptors, respectively, which in turn transmit itch signals to the brain, leading to scratching