CONCLUD

Interestingly, mRNA expression of TRPM2 channel, which is known to contribute to inflammatory and neuropathic pain (3), was up-regulated in the bladders of both WT and TRPA1-KO mice with LPS-instillation, suggesting that TRPM2 channel would be further explored as a target for bladder inflammatory pain disorders.

Concluding message

TRPA1-KO mice showed attenuated bladder hyperactivity induced by LPS-instillation, but similar inflammatory changes in the bladder as WT mice. These results suggest that TRPA1 is involved in bladder hyperalgesia induced by bladder inflammation, but not in inflammation itself.
Figure 1. Representative tracings of intravesical pressure in WT and TRPA1-KO mice at 24 hours after LPS-instillation under a decerebrate unanesthetised condition

Figure 2. Representative images of the bladder in saline-instilled (A, D) and LPS-instilled (B, E) WT mice and LPS-instilled TRPA1-KO mouse (C, F) at 24 hours after instillation. Comparison between groups of the number of infiltrating inflammatory cells in the suburothelial layer of the bladder (N = 7 in each group) (G)

Scale bar, A-C: 200µm. D-F: 100µm. The square area in each upper panel (A-C) was corresponding to each lower panel (D-F).

*p<0.05, *** p<0.001: significant differences from WT mice with saline-instillation. No significant differences were found between LPS-treated WT and TRPA1-KO mice (Tukey test).

Figure 3. mRNA expressions of TRP channels in the bladder (A, B) and L6 DRG (C) of WT and TRPA1-KO mice at 24 hours after saline- or LPS-instillation (N = 5 in each group)

*p<0.05, ** p<0.01: significant differences between WT mice with saline-instillation and WT mice with LPS-instillation (unpaired t-test).

References
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