Correction

Correction: Sung et al., “Swedish Nerve Growth Factor Mutation (NGFR100W) Defines a Role for TrkA and p75NTR in Nociception”

In the article “Swedish Nerve Growth Factor Mutation (NGFR100W) Defines a Role for TrkA and p75NTR in Nociception” by Kijung Sung, Luiz F. Ferrari, Wanlin Yang, ChiHye Chung, Xiaobei Zhao, Yingli Gu, Suzhen Lin, Kai Zhang, Bianxiao Cui, Matthew L. Pearn, Michael T. Maloney, William C. Mobley, Jon D. Levine, and Chengbiao Wu, which appeared on pages 3394–3413 of the April 4, 2018 issue:

- (1) The authors discovered an error in the Introduction on page 3396 (left column: lines 20–21). The corrected text is as follows: “In contrast, signaling through p75 appears to be necessary for acute sensitization.”
- (2) The authors discovered an error in the Materials and Methods on page 3396 (right column: lines 45–46). The corrected text is as follows: “Administration of wtNGF, NGF^{R100W}, prostaglandin E2 (PGE2), and inhibitors to adult rats.”
- (3) The authors discovered an error in the Materials and Methods starting from the last paragraph of the right column on page 3396 and end at the second paragraph of the left column on page 3397. The authors request to delete the following text: “To attenuate the expression of TrkA, the antisense (AS) sequence 5'-CAT CAA CGA AGT CAC CAG ACC G-3' was directed against a unique sequence of rat TrkA (Alvarez and Levine, 2014). The corresponding GenBank accession number and ODN position within the cDNA sequence are M85214 and 121–142, respectively. TheMMODN sequence 5'-CAA CAT CGA AGT GAC GAG ACC G-3' corresponds to the TrkA subunit AS sequence with four bases mismatched (denoted by boldface letters). We have shown previously that intrathecal administration of TrkA AS completely eliminated NGF-induced mechanical hyperalgesia (Malik-Hall et al., 2005). For reducing the expression of p75NTR, the AS sequence (5'-ACC TGC CCT CCT CAT TGC A-3') was directed against a segment of the rat p75NTR mRNA that spans the start codon (Barrett et al., 2016). The MM sequence for p75NTR was: 5'-CTC CCA CTC GTC ATT CGA C-3'. All AS and MM primers were from Invitrogen. Oligos were administered by intrathecal injection. Rats were briefly anesthetized with 2.5% isoflurane in 97.5% of O₂. Then, a 30-gauge hypodermic needle was inserted into the subarachnoid space on the midline between the L4 and L5 vertebrae and the injection of AS or MM or ASMM oligos performed (80 g/20 L). Intrathecal site of injection was confirmed by checking for a sudden flicking of the tail (Ferrari et al., 2010, 2013, 2015a,b). The injection scheme and experimental design is illustrated in Figure 7B.”
- (4) The authors discovered an error in their Results on page 3404 (line 14). The corrected text is as follows: Figure 6H.
- (5) The authors discovered an error in their Discussion on page 3408 (lines 16–40). The corrected text is as follows: “Consistent with previous results (Covaceuszach et al., 2010; Capsoni et al., 2011), we confirmed that, whereas NGF^{R100W} binding and activation of TrkA is robust, binding and signaling through p75^{NTR} is essentially absent. Important to its ability to prime nociceptors, NGF^{R100W} was internalized at axonal tips of DRG sensory neurons and traveled toward the cell body at the speed and with the velocity characteristic of wtNGF. Given properties in common with wtNGF, it is unclear why NGF^{R100W} failed to induce acute sensitization. Possibilities include differences with respect to wtNGF in the structure of the NGF^{R100W}/TrkA complex, the structure of the complex in signaling endosomes, or the downstream partners recruited to these complexes. Furthermore, it is hard to exclude the possibility that TrkA downstream signaling events implicated in pain could be positively affected by crosstalk between p75^{NTR} and TrkA, as suggested by only partial PLC γ activation by NGF^{R100W}. This suggests the possibility that TrkA downstream events that support trophic functions are retained in NGF^{R100W}, whereas p75^{NTR} downstream and some TrkA downstream events that affect pain threshold are reduced in NGF^{R100W}. In any case, it is apparent that differences must exist for the signaling events that subserve TrkA trophic functions and its acute effects on nociceptor sensitization. Further studies to decipher the basis for these differences will benefit from the ability to compare the signaling properties of NGF^{R100W}/TrkA with wtNGF/TrkA.”

The authors sincerely apologize for these oversights. These changes do not affect any other figures or results in the manuscript, and do not affect any conclusions or interpretations in the manuscript.

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