Correction for Volume 16, p. 182

Genome Res. 2006 16: 557

Errata

Genome Research 13: 781–793 (2003)

Complex evolution of 7E olfactory receptor genes in segmental duplications

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Using a newer human genome assembly (May 2004, hg17), a better alignment and more stringent criteria, the authors find far fewer putative instances of ectopic exchanges involving the 7E subfamily of odorant receptor pseudogenes than originally reported (Newman and Trask 2003). Each of the three legs of support for this phenomenon is weakened. First, the shift in best-matching partners in Figure 5, and associated percent identities, is an artifact of the August 2001 genome assembly (hg 8). Second, the original alignment did not optimally position some frame-shifting mutations in these genes. An improved alignment of genes from the newest assembly (coordinates in Supplemental Table A; alignment in Supplemental Data) reveals that all but six of the 35 taxa in the B clade share a 4-nucleotide frameshift mutation in TM3 and a stop mutation (TTA→TGA) in TM6 (Supplemental Fig. A). The TM6 mutation is also shared by 24 of 46 taxa in clade A. Although the presence of the TM6 mutation in some but not all members of both clades is consistent with a past gene-conversion event, this pattern also could have resulted from two independent, identical mutations. Finally, use of GeneConv (Sawyer 1989; http://www.math.wustl.edu/~sawyer/geneconv/) on various subsets of 7E sequences using the updated and improved alignment implicates only a few genes in putative conversion events that are statistically significant after Bonferroni correction for multiple tests. Thus, while 7E-containing segmental duplications might be subject to ectopic exchange events (e.g., Giglio et al. 2001), statistically supported signatures of these events are not prevalent in the 7E pseudogene sequences. The authors regret any confusion these artifacts in their analysis might have caused, and thank Eleanor Williams for bringing them to their attention.

References

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Newman, T. and Trask, B.J. 2003. Complex evolution of 7E olfactory receptor genes in segmental duplications. *Genome Res.* **13:** 781–793.
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Evolution of alternative splicing after gene duplication

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The authors inadvertently failed to give precedence to the recent paper of N.M. Kopelman, D. Lancet, and I. Yanai (2005), "Alternative splicing and gene duplication are inversely correlated evolutionary mechanisms," *Nat. Genet.* **37:** 588–589, which reports equivalent findings. Namely, that an inverse correlation exists between the size of a gene's family and its use of alternatively spliced isoforms. The authors sincerely apologize to those concerned for this oversight.