Corrigendum: Expression profiling of medulloblastoma: PDGFRA and the RAS/MAPK pathway as therapeutic targets for metastatic disease

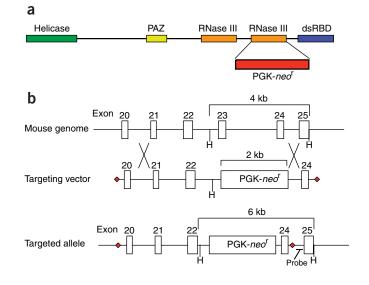
T J MacDonald, K M Brown, B LaFleur, K Peterson, C Lawlor, Y Chen, R J Packer, P Cogen & D A Stephan *Nat. Genet.* 29, 143–152 (2001)

The annotation for the Affymetrix G110 probe set 1770 that we used was incorrect. Although the annotation specifies that the transcript for PDGFR- α is being ascertained, the true specificity of the probe set is for the PDGFR- β isoform. The ligand for both receptor isoforms is identical. The functional validation of the PDGFR signaling pathway, described in our article, used specific neutralizing antibodies against PDGFR- α as well as downstream small molecule inhibitors, and it implicates this entire cascade. The PDGFR- β isoform may be more relevant in the metastatic process, but this does not discount the proven biologic role of PDGFR- α and downstream effectors in metastatic medulloblastoma.

Corrigendum: Dicer is essential for mouse development

E Bernstein, S Y Kim, M A Carmell, E P Murchison, H Alcorn, M Z Li, A A Mills, S J Elledge, K V Anderson & G J Hannon *Nat. Genet.* 35, 215–217 (2003)

Figure 1a incorrectly showed that the disruption targeted the first RNase III domain. This error also occurred throughout the text. In fact, the construct targets the second RNase III domain, which contains both canonical and noncanonical active sites. Because both RNase III domains are thought to pair intramolecularly to form an active Dicer, the construct that targets the second RNase III domain is also predicted to be a null allele. This is confirmed by the mutant shown in **Figure 1d**, in which sequences removed from human Dicer indeed corresponded to those removed from the mouse (correctly listed as residues 1,686–1,728 but incorrectly attributed to the first RNase III domain). Finally, since the targeting construct was generated, ENSEMBL has updated the mouse *Dicer1* gene prediction, renumbering the exons. In the current release, the exon targeted in our disruption is exon 23. We apologize for this error but note that it does not alter the data presented, its interpretations or the conclusions of the paper. Corrected versions of **Figure 1a** and **b** appear below.



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