CORRIGENDUM

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Corrigendum: Comprehensive genomic characterization defines human glioblastoma genes and core pathways

The Cancer Genome Atlas Research Network

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In this Article, we reported somatic mutations in the human ERBB2 gene in 7 of 91 cases analysed by capillary DNA sequencing and validated by mass spectrometric genotyping. Further analysis of these cases has revealed that the mutations were present only in the whole-genome amplified tumour DNA used for the study but not in the unamplified tumour DNA (see ref. 1 for more details). The reported mutations are likely to be artefacts of whole-genome amplification, because the ERBB2 mutations in our Article were not validated in unamplified DNA. The validity of recurrent mutations in other genes besides ERBB2 that were reported to be significantly mutated in our Article (namely TP53, PTEN, NF1, EGFR, RB1, PIK3R1 and PIK3CA) has been confirmed by The Cancer Genome Atlas Research Network using unamplified DNA (ref. 2).

1. Greulich, H, *et al.* Functional analysis of receptor tyrosine kinase mutations in lung cancer identifies oncogenic extracellular domain mutations of ERBB2. *Proc. Natl Acad. Sci. USA* **109,** 14476–14481 (2012).
2. The Cancer Genome Atlas Research Network. The somatic genomic landscape of glioblastoma. *Cell* (submitted).