CORRIGENDUM

A mutation in the 3′-UTR of the HDAC6 gene abolishing the post-transcriptional regulation mediated by hsa-miR-433 is linked to a new form of dominant X-linked chondrodysplasia

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The authors of the above paper would like to apologize for an error in Figure 6D. In the original version the lettering in the figure was truncated. The corrected figure appears below:
Figure 6. Fibroblasts analysis of patient III16. (A) Patient III16 at the age of 3.5 years showing the strikingly much shorter leg when compared with the right leg; the same growth difference can also be seen on the left arm, although less obviously. (B) X chromosome inactivation analysis was performed on DNA extracted from fibroblasts obtained from skin biopsies of the right (B1) and left (B2) arms of the patient, and on DNA extracted from lymphocytes from control females without (B3) and with (B4) a skewed X chromosome inactivation pattern. 600ng DNA was digested with restriction enzymes HpaII and CfoI (lower panels) or not digested (upper panels) before PCR was performed to amplify a fragment containing the polymorphic (CAG)n repeat of the HUMARA gene. Size analysis of the PCR products was performed after fractionation on an ABI3130XL gene analyser (Applera). (C) RNA was extracted from the left arm- and right arm-derived fibroblasts and retrotranscribed to cDNA. Exon 29 was amplified by PCR and sequenced. The boxes indicate the location of the variant base in the HDAC6 3'-UTR. Only the 'A' (normal) allele is expressed in the right arm, whereas the 'T' allele (variant) is expressed in \( \approx 30\% \) of cells. (D) Segregation of the (CAG)n repeat HUMARA alleles in the family under study. Only the individuals for whom X chromosome inactivation analysis has been performed are represented. Allele 190 which is on the inactivated X chromosome in the lymphocytes of heterozygote carriers in this family is underlined. Deduced genotype is shown in parentheses.