

CORRIGENDA

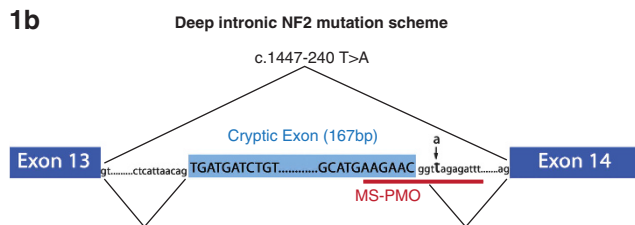
## *In vitro* antisense therapeutics for a deep intronic mutation causing Neurofibromatosis type 2

Elisabeth Castellanos, Imma Rosas, Ares Solanes, Isabel Bielsa, Conxi Lázaro, Cristina Carrato, Cristina Hostalot, Pepe Prades, Francesc Roca-Ribas, Ignacio Blanco and Eduard Serra on behalf of the NF2 Multidisciplinary Clinics HUGTiP-ICO-IMPPC

*European Journal of Human Genetics* (2014) 22, 153; doi:10.1038/ejhg.2013.224

**Correction to:** *European Journal of Human Genetics* (2013) 21, 769–773; doi:10.1038/ejhg.2012.261; published online 28 November 2012

Post publication the authors realized that they had made some errors in their article for which they would like to apologise. g.74409 T>A the mutation at the genomic level should be termed: g.74408 T>A, and the sequence of the scheme depicted in Figure 1b, contains a couple of single nucleotide changes. A revised figure is shown below.



**Figure 1 Deep intronic NF2 mutation description.** (a) Analysis of patient fibroblasts showed a proportion of NF2 transcripts containing the inclusion of a cryptic exon (NF2 CEI) compared with the WT NF2 mRNA (NF2 WT) (upper panel). Forward sequence of cryptic exon inclusion is shown (bottom panel). (b) Schematic representation of the identified NF2 deep intronic mutation and MS-PMO location. Constitutive and cryptic exons are represented by dark and light gray boxes, respectively. The boundaries of the cryptic inserted exon are shown in uppercase; flanking intronic sequences are shown in lowercase. MS-PMO sequence is underlined. Mutated nucleotide is shown in bold and the nucleotide change is indicated by an arrow.

## Return of whole-genome sequencing results in paediatric research: a statement of the P<sup>3</sup>G international paediatrics platform

Bartha Maria Knoppers, Denise Avard, Karine Sénécal and Ma'n H Zawati along with the P<sup>3</sup>G International Paediatrics Platform members

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The authors have added an acknowledgement to their paper since online publication:

This work was supported by grants from the Canadian Institutes of Health Research and the Terry Fox Foundation (TFF-105266), Genome Canada and the Canadian Institutes of Health Research, Finding of Rare Disease Genes in Canada (FORGE), the Maternal Infant and Youth Research Network (MICYRN),

the Network of Applied Genetic Medicine of Québec (RMGA) and the Fonds de recherche en santé du Québec (FRSQ).

The corrected article appears in this issue and the HTML and online PDF now carry the additional text. The authors would like to apologise for this omission.

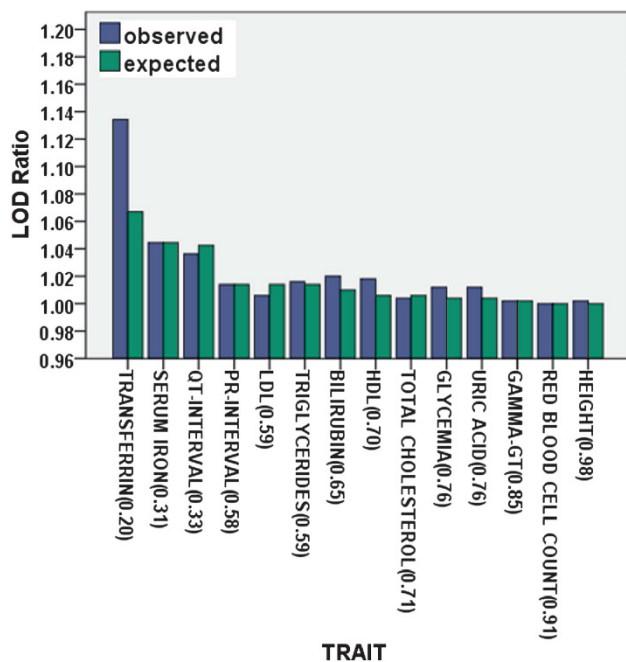
# SHAVE: shrinkage estimator measured for multiple visits increases power in GWAS of quantitative traits

Osorio D Meirelles, Jun Ding, Toshiko Tanaka, Serena Sanna, Hsih-Te Yang, Dawood B Dudekula, Francesco Cucca, Luigi Ferrucci, Goncalo Abecasis and David Schlessinger

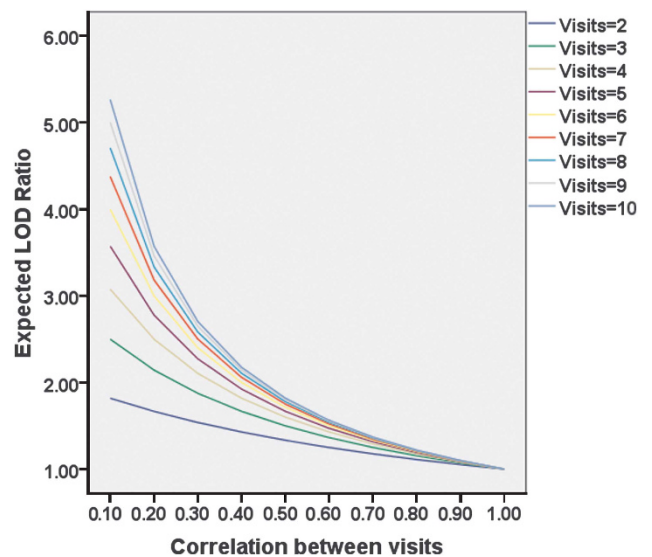
*European Journal of Human Genetics* (2014) **22**, 154; doi:10.1038/ejhg.2013.218

**Correction to:** *European Journal of Human Genetics* (2013) **21**, 673–679; doi:10.1038/ejhg.2012.215; published online 24 October 2012.

It has come to the attention of the authors that the legends for Figures 3 and 4 had been transposed. The figures with their corresponding legends appear below.



**Figure 3** Observed and expected LOD ratio for SHAVE and Average for top SNPs from meta-analysis for a subset of individuals in which all individuals had visit 1 and a randomly chosen 50% of visit 2 cases were selected among the same individuals.



**Figure 4** Expected LOD ratio between average and single visit for hypothetical datasets in which all individuals had  $k$  visits ranging from 2 to 10.