

# Corrigendum

## Biallelic inactivation of *REV7* is associated with Fanconi anemia

Dominique Bluteau, Julien Masliah-Planchon, Connor Clairmont, Alix Rousseau, Raphael Ceccaldi, Catherine Dubois d'Enghien, Olivier Bluteau, Wendy Cucchini, Stéphanie Gachet, Régis Peffault de Latour, Thierry Leblanc, Gérard Socié, André Baruchel, Dominique Stoppa-Lyonnet, Alan D. D'Andrea, and Jean Soulier

Original citation: *J Clin Invest*. 2016;126(9):3580–3584. <https://doi.org/10.1172/JCI88010>.

Citation for this corrigendum: *J Clin Invest*. 2017;127(3):1117. <https://doi.org/10.1172/JCI92946>.

The nomenclature for the *REV7* mutation was incorrectly noted in two sentences in the second paragraph of Results and Discussion and in Figure 1H. The correct sentences and figure part are below.

Whole exome sequencing (WES) on genomic DNA from the EGF123 proband identified a homozygous *REV7* variant, c.254T>A.

The c.254T>A *REV7* is a variant based on a survey of publicly accessible variant databases.

In addition, the fourth sentence of the Abstract was incorrect. The correct sentence is below.

Patient-derived cells demonstrated an extended FA phenotype, which included increased chromosome breaks and G<sub>2</sub>/M accumulation upon exposure to DNA crosslinking agents,  $\gamma$ H2AX and 53BP1 foci accumulation, and enhanced p53/p21 activation relative to cells derived from healthy subjects.

The authors regret the errors.

