

CORRECTION

Correction: Emerging roles of the single EF-hand Ca^{2+} sensor tescalcin in the regulation of gene expression, cell growth and differentiation

Ksenia G. Kolobynina, Valeria V. Solovyova, Konstantin Levay, Albert A. Rizvanov and Vladlen Z. Slepak

There was an error published in *J. Cell Sci.* **129**, 3533–3540.

Incorrect language was used in the Commentary that concerns the apparent controversy between the results of tescalcin knockdown in hematopoietic precursor cell lines and its gene knockout in mice reported by Ukarapong et al. (2012). In the Commentary, the wording is as follows: “To explain the conflicting data, the authors [Ukarapong et al.] speculate that CHP1 and/or CHP2 could compensate for the lack of tescalcin, or that their genetic knockout was incomplete and allowed for the expression of a partial protein (Ukarapong et al., 2012)”. This gives the impression that Ukarapong et al. themselves argued about the incomplete knockout.

The wording should read: “To explain the conflicting data, the authors [Ukarapong et al.] speculate that CHP1 and/or CHP2 could compensate for the lack of tescalcin (Ukarapong et al., 2012). Another possibility can be associated with the presence of a transcript corresponding to exons 1, 2, 7 and 8 of the TESC gene in the knockout mouse tissues detected by Ukarapong et al. Although unlikely, this finding leaves a formal possibility for this mRNA to be translated into a product with residual functional activity”.

The authors of the Commentary apologize to Ukarapong et al. and to the readers of Journal of Cell Science for any confusion that this error may have caused.

Reference

Ukarapong, S., Bao, Y., Perera, E. M. and Berkovitz, G. D. (2012). Megakaryocyte development is normal in mice with targeted disruption of Tescalcin. *Exp. Cell Res.* **318**, 662–669.