ERRATUM Open Access



## Erratum to: Repression of chimeric transcripts emanating from endogenous retrotransposons by a sequence-specific transcription factor

Ka Sin Mak, Jon Burdach, Laura J. Norton, Richard CM Pearson, Merlin Crossley\* and Alister PW Funnell

In the study [1] a gel depicted in Fig. 2a was labelled in a way which suggests that the sample comes from a  $Klf3^{-/-}$  knockout mouse. In fact, this sample comes from a  $Klf3^{-/-}$ ,  $Klf8^{\rm genetrap}$  double mutant animal. The Klf8 genotype was not indicated as authors felt that it was not relevant for the conclusions of this paper; however, all authors now acknowledge that this information should have been included. Importantly, an equivalent result from single  $Klf3^{-/-}$  knockout mice is included and confirmed in the RNA-seq results presented in Figure 6 of the original article [1].

All other data described in the article were obtained from the  $Klf3^{-/-}$  single knockout mice, and as such the conclusions of the article remain unchanged. It is also critical to note that all other results in the article could not have been obtained from the double mutant mice, because Klf3,Klf8 deficient animals die *in utero* (as reported by the authors in [2]).

Figure 2 with the correct legend is published in this Erratum.

The authors apologize for this omission and any confusion and inconvenience it may have caused.

Received: 5 May 2016 Accepted: 5 May 2016 Published online: 03 June 2016

## References

- Mak KS, Burdach J, Norton LJ, Pearson RCM, Crossley M, Funnell APW. Repression of chimeric transcripts emanating from endogenous retrotransposons by a sequence-specific 35 transcription factor. Genome Biol. 2014;15:R58.
- Funnell APW, Mak KS, Twine NA, Pelka GJ, Norton LJ, Radziewic T, et al. Generation of Mice Deficient in both KLF3/BKLF and KLF8 Reveals a Genetic Interaction and a Role for These Factors in Embryonic Globin Gene Silencing. Mol Cell Biol. 2013;33(15):2976–87.

## Submit your next manuscript to BioMed Central and we will help you at every step:

- We accept pre-submission inquiries
- Our selector tool helps you to find the most relevant journal
- We provide round the clock customer support
- Convenient online submission
- · Thorough peer review
- Inclusion in PubMed and all major indexing services
- Maximum visibility for your research

Submit your manuscript at www.biomedcentral.com/submit





© 2016 Mak et al. **Open Access** This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.

<sup>\*</sup> Correspondence: m.crossley@unsw.edu.au School of Biotechnology and Biomolecular Sciences, University of New South Wales, Kensington, NSW 2052, Australia

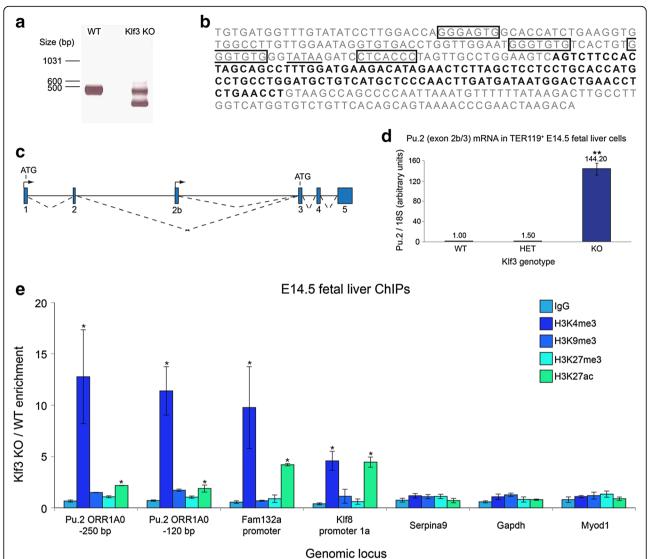


Fig. 2 A novel, internal *Pu.1* promoter resides within an *ORR1A0* LTR element and is repressed by KLF3. (a) RNA from *Klf3*+/+ (WT) and *Klf3*-/-, *Klf8*<sup>genetrap</sup> TER119+ fetal liver cells was subjected to 5' RACE using a reverse primer specific for exon 3 of *Pu.1* and analyzed by agarose gel electrophoresis. The smaller band from a *Klf3*KO animal was sequenced and found to contain a novel exon (exon 2b). (b) The sequence of the *ORR1A0* LTR, in which *Pu.1* exon 2b is shown in bold. Sequences which fit the KLF binding consensus 5'-NCN CNC CCN-3' are boxed, and the TATA box at –30 is underlined. (c) Schematic of the murine *Pu.1* locus showing the position of exon 2b. Exons are represented by blue boxes, transcription start sites by arrowheads and splicing events by broken lines. Start points of translation (ATGs) for the two alternative transcripts are also shown. (d) Real-time RT-PCR quantification revealing that transcripts containing exon 2b spliced to exon 3 of *Pu.1* (that is, *Pu.2* transcripts) are upregulated in *Klf3*-/- TER119+ E14.5 fetal liver cells compared to *Klf3*+/- (HET) and *Klf3*+/- values have been normalized to *185* rRNA and the *Klf3*+/- sample has been set to 1.0. *n* = 3 for each genotype.

\*\*\*, P <0.005 compared to both *Klf3*+/- and *Klf3*+/- (Student's two-tailedt-test). (e) ChIPs were performed on *Klf3*+/- and *Klf3*-/- E14.5 fetal livers (*n* = 2 or 3 of each genotype per IP). Data are represented as the fold-change enrichment in *Klf3*-/- cells compared to *Klf3*+/+. The *Fam132a* and *Klf8* promoters have been included as positive controls while *Serpina9*, *Gapdh*, and *MyoD* are negative control regions. \*\*\*, P <0.05 compared to *Gapdh* (Student's one-tailed *t*-test). In (d and e), error bars represent standard error of the mean