cells

MDPI

Correction

Correction: Furlan, G. and Galupa R. Mechanisms of Choice in X-Chromosome Inactivation. *Cells* 2022, *11*, 535

Giulia Furlan [1,2,*,†] and Rafael Galupa [3,*,†]

1 Wellcome Trust/Cancer Research UK Gurdon Institute, University of Cambridge, Tennis Court Road, Cambridge CB2 1QN, UK
2 Department of Genetics, University of Cambridge, Downing Street, Cambridge CB2 3EH, UK
3 Developmental Biology Unit, European Molecular Biology Laboratory, 69117 Heidelberg, Germany
* Correspondence: gf329@cam.ac.uk (G.F.); rafael.galupa@embl.de (R.G.)
† These authors contributed equally to this work.

The authors wish to make the following changes to their paper [1]. In the original publication, there was a mistake uploading the figures, and Figures 1–3 as published do not correspond to the updated versions after peer review. In particular, Figure 1 needed to be corrected regarding the transcriptional status of the X chromosomes during the preimplantation stages. Figures 1–3 should be changed to:

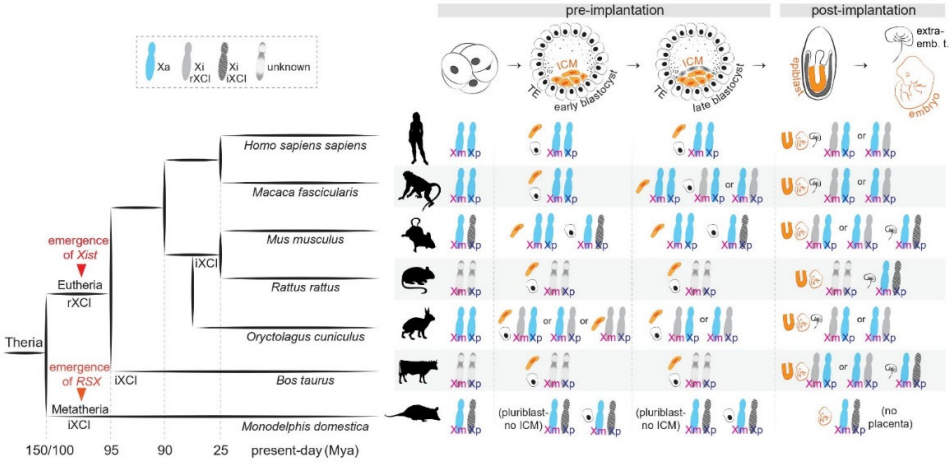

Figure 1. X-chromosome inactivation across species. (**Left**): Phylogenetic tree indicating the evolution of random and imprinted XCI and the emergence of long non-coding RNAs *Xist* and *RSX* in Theria. (**Right**): X-chromosome inactivation dynamics across development in representative species.

Citation: Furlan, G.; Galupa, R. Correction: Furlan, G. and Galupa R. Mechanisms of Choice in X-Chromosome Inactivation. *Cells* 2022, *11*, 535. *Cells* **2023**, *12*, 950. https://doi.org/10.3390/cells12060950

Received: 3 November 2022
Accepted: 4 November 2022
Published: 21 March 2023

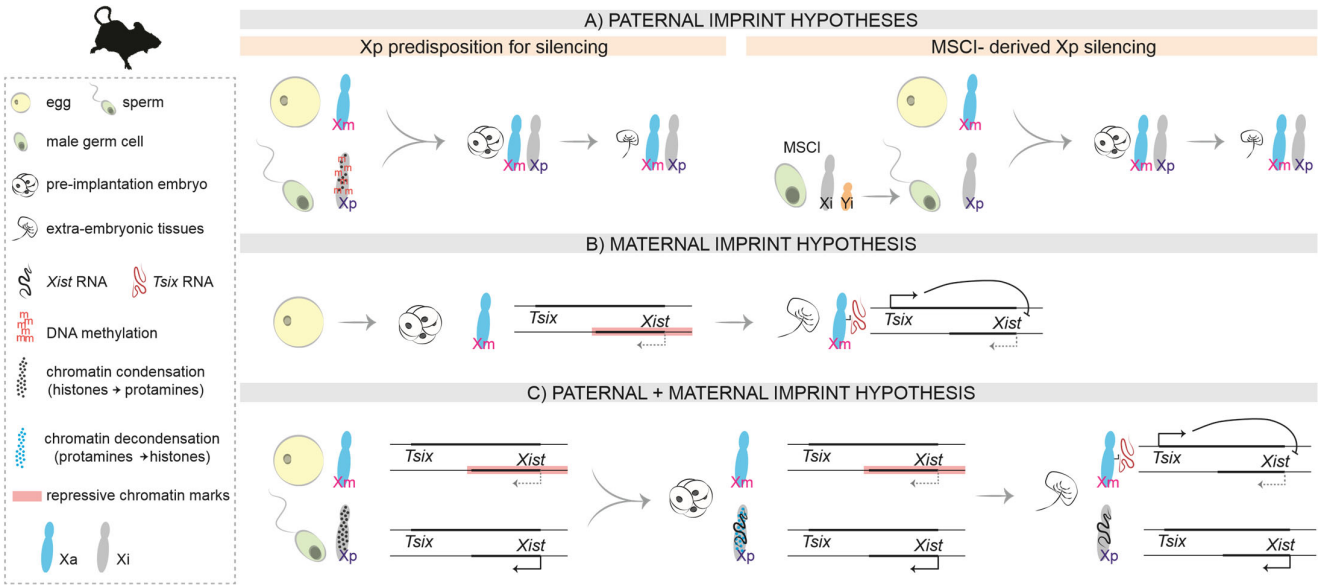

Figure 2. Hypotheses on the molecular nature of the imprint in mice. (**A**). Paternal imprint: The Xp inherits a predisposition for silencing from its life cycle in the male. (**B**). Maternal imprint: In the preimplantation embryo, repressive chromatin marks on the Xm (including the *Xist* promoter region) prevent *Xist* expression on the Xm. In the extra-embryonic tissues of the post-implantation embryo, *Tsix* expression prevents *Xist* upregulation in *cis*. (**C**). Paternal and maternal imprint: A combination of both hypotheses, also considering the different chromatin condensation states of the Xp in the sperm and in the paternal pronucleus after fertilization.

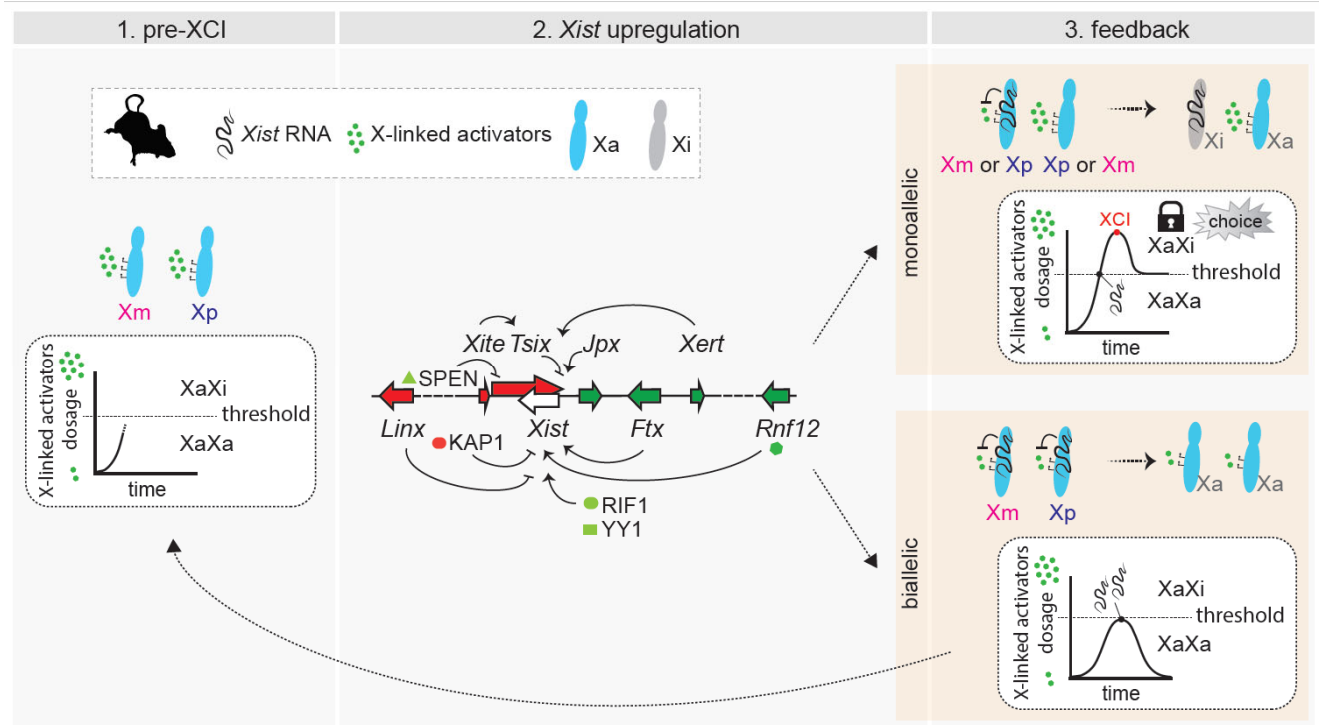

Figure 3. Dynamic model of choice during random XCI. (**Left**): Pre-XCI status. Both X chromosomes are active and transcribe X-linked genes. The dose of X-linked activators increases towards the threshold necessary for productive *Xist* upregulation. (**Middle**): Biallelic X-chromosome transcription allows the cell to reach the threshold for *Xist* activation. X-linked and autosomal *cis* and *trans* positive

and negative regulators influence the initiation of *Xist* upregulation, which can occur on a single X chromosome or on both of them. Only factors and loci discussed in the text have been included in the figure. (**Right** (**top**)): In cells that have upregulated *Xist* monoallelically, X-wide *cis*-silencing triggered by *Xist* RNA causes a drop in the level of activators, preventing the second chromosome from upregulating *Xist*. The choice is locked in. Monoallelic *Xist* expression (and *cis*-silencing) has to be sustained through enough dosage of activators and/or feedback mechanisms. (**Right** (**bottom**)): In cells that have upregulated *Xist* biallelically, excess *Xist* expression triggers rapid downregulation of X-linked activators on both X chromosomes, and this drop in levels below the threshold causes *Xist* expression to switch off. Both X chromosomes remain active, and the process has to start again.

The authors apologize for any inconvenience caused and state that the scientific conclusions are unaffected. This correction was approved by the academic editor. The original publication has also been updated.

Conflicts of Interest: The authors declare no conflict of interest.

Reference

1. Furlan, G.; Galupa, R. Mechanisms of Choice in X-Chromosome Inactivation. *Cells* **2022**, *11*, 535. [CrossRef] [PubMed]