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Morita, Shuhei Villalta, S Armando Feldman, Hannah C et al.

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Targeting ABL-IRE1α Signaling Spares ER-Stressed Pancreatic β Cells to Reverse Autoimmune Diabetes

Shuhei Morita, S. Armando Villalta, Hannah C. Feldman, Ames C. Register, Wendy Rosenthal, Ingeborg T. Hoffmann-Petersen, Morvarid Mehdizadeh, Rajarshi Ghosh, Likun Wang, Kevin Colon-Negron, Rosa Meza-Acevedo, Bradley J. Backes, Dustin J. Maly,* Jeffrey A. Bluestone,* and Feroz R. Papa*
*Correspondence: djmaly@uw.edu (D.J.M.), jeff.bluestone@ucsf.edu (J.A.B.), frpapa@medicine.ucsf.edu (F.R.P.)
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In the originally published version of this article, the immunoblot image of the HDAC1 nuclear extract protein control in Figure 3I was incorrectly cropped such that it included one extraneous lane. The corrected and original versions of Figure 3I are shown here. Furthermore, in the Discussion, after the sentence "Such compensatory, dysregulated UPR effects may be general as Perk deletion, likewise, hyperactivates IRE1 α in β cells, which suffer early apoptosis, leading to postnatal diabetes," we incorrectly cited Harding, H.P., and Ron, D. (2002). Endoplasmic reticulum stress and the development of diabetes: a review. Diabetes 51, S455–S461. The correct citation is: Harding, H.P., Zeng, H., Zhang, Y., Jungries, R., Chung, P., Plesken, H., Sabatini, D.D., and Ron, D. (2001). Diabetes mellitus and exocrine pancreatic dysfunction in perk-/- mice reveals a role for translational control in secretory cell survival. Molecular Cell 7, 1153–1163. The authors apologize for any confusion these errors may have caused.

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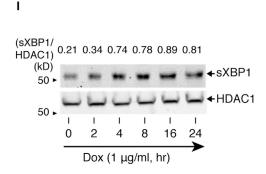


Figure 3I. ABL Family Tyrosine Kinases Are Necessary and Sufficient for Driving T-UPR-Mediated Apoptosis through IRE1a (corrected)

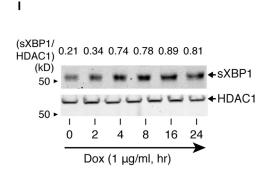


Figure 3I. ABL Family Tyrosine Kinases Are Necessary and Sufficient for Driving T-UPR-Mediated Apoptosis through IRE1α (original)

