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Glucose-induced β cell production of IL-1 β contributes to glucotoxicity in human pancreatic islets

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Corrigendum

Original citation: J Clin Invest. 2002;110(6):851–860. https://doi.org/10.1172/JCl15318 Citation for this corrigendum: J Clin Invest. 2017;127(4):1589. https://doi.org/10.1172/JCl92172 The editors recently became aware that three images in Figure 4F of this article are duplicated in a 2002 Proceedings of the National Academy of Science of the United States of America (PNAS) publication by this group (1). The specific panels of Figure 4F that were duplicated are the Fas-stained islets cultured in 5.5 mM glucose + IL-1 β , the insulin-stained islets cultured in 5.5 mM glucose, and the insulin-stained islets cultured in 5.5 mM glucose + IL-1 β . The images appear in the PNAS publication as representing different treatment conditions. The authors were able to provide the original source data for both the JCl and PNAS figures. They determined that the correct images appear in the PNAS paper; however, the same images were inadvertently incorporated into the JCl paper due to similarities in the blinded code file numbers assigned to the correct images. The authors also determined that the incorrect image was used for the Fas/insulin double-stained islets cultured in 33.3 mM glucose. The corrected panel appears below. The authors regret the errors.

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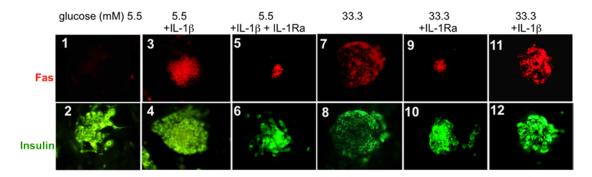
Glucose-induced β cell production of IL-1β contributes to glucotoxicity in human pancreatic islets

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1. Maedler K, et al. FLIP switches Fas-mediated glucose signaling in human pancreatic beta cells from apoptosis to cell replication. *Proc Natl Acad Sci U S A*. 2002;99(12):8236–8241.