AUTHOR CORRECTION

Author Correction: Beta cell connectivity in pancreatic islets: a type 2 diabetes target?

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The original version of this article unfortunately contained a mistake. Legends of Figs. 1 and 2 were interchanged. The correct versions are given below.

The original article can be found online at <https://doi.org/10.1007/s00018-014-1755-4>.

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Fig. 1 Imaging and mapping beta cell network topology. (Above) Functional multicellular Ca^{2+} imaging is used to monitor the large-scale organization of glucose-induced population dynamics (above, left). By subjecting the resulting traces (from ~ 50–100 individual cells per islet) to correlation analyses, cells with coordinated activity can be identified and a functional connectivity map plotted based upon position within the imaged field (x–y) (above, right). Scale-free connection distributions are typified by a minority of cells that host the majority of connections (nodes), while maintaining streamlined information flow due to a short pathlength. Although robust in the face of random attack, they are prone to collapse following a targeted attack (below, left). By contrast, nonscale-free networks (e.g., random or lattice) may not efficiently propagate signals due to a long pathlength, and random attacks significantly reduce capacity (below, right)

Fig. 2 Schematic showing single cell and population-level beta cell signaling. At the molecular level, glucose is transported into the beta cell before undergoing glycolysis to increase the ratio of free cytosolic ATP:ADP. This closes K_{ATP} channels, leading to opening of VDCC, Ca^{2+} influx, and Ca^{2+} -dependent exocytosis. At the population-level, beta cell dynamics are further dictated by signaling circuits involving paracrine, juxtacrine, autocrine, electrotonic (GJ), neural and ciliary communications