Correction to: Identification of an intracellular metabolic signature impairing beta cell function in the rat beta cell line INS-1E and human islets

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As part of an institutional investigation by University of Bremen, the work carried out by Kathrin Maedler's laboratory has been reviewed. The investigation found no intentional or grossly negligent misrepresentation but owing to file storage problems the authors cannot be certain of the origin of the β -actin lane of the western blot shown in Fig. 4b of the above paper. The authors no longer have access to the β -actin blot source file from 2010 so the blot is corrected here using lanes from one of the other two replicates performed at the time of the original experiment. The densitometry analysis in Fig. 4c is updated to reflect only data from the two replicates and shows the same results as that published originally. This correction does not affect the results or conclusion of the paper.

The online version of the original article can be found at <https://doi.org/10.1007/s00125-011-2249-7>

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Fig. 4 Increased ERK1/2 activity during acute and chronic high glucose (glc) treatment and after PPP inhibition at low extracellular glucose concentrations. Western blot analyses of p-ERK1/2 of human islets (a) exposed to 5.5, 11.1 and 33.3 mmol/l glucose during 72 h culture and (b) cultured at 5.5 and 22.2 mmol/l glucose and at 5.5 mmol/l glucose+

500 μ mol/l 6-AN for 45 min and 72 h. A representative blot is shown. Densitometry analyses of bands normalised to tubulin (c) from two independent experiments from two organ donors. Data are mean $\pm$ SE. 6-AN, 6-Aminonicotinamide; PPP, pentose phosphate pathway