CORRIGENDUM

INSIDE LAB INVEST

Laboratory Investigation (2007) **87**, 515. doi:10.1038/labinvest.3700557

Correction to: *Laboratory Investigation* (2007) **87**, 94–95. doi:10.1038/labinvest.3700510

Following the publication of the editorial: *β -Cell-specific aggregation of cellular prion protein*, the editor wishes to make the following revision:

Cellular prion protein (PrP^C) is expressed abundantly in neuronal and several other tissues and its function is unknown. In this issue, Strom *et al* (p 139) hypothesized that PrP^C expression in the endocrine pancreas is related to the regulation of glucose homeostasis. By using a model of spontaneous type 1 diabetes and a model of β -cell adaptation to chronic glucose infusion, these authors showed a distinctive accumulation of cytosolic PrP^C inclusions in a subset of insulin-producing β -cells. The frequency of β -cells with PrP^C inclusions was three-fold greater in diabetes-prone rats, suggesting that PrP^C accumulation plays a role in β -cell dysfunction. These observations open new avenues for the study of diabetes pathogenesis. One of the most pressing questions to address is the relationship between PrP^C accumulation and the autoimmune process; specifically, PrP^C

accumulation could somehow promote the disease, with β -cells containing PrP^C aggregates initiating the autoimmune process by release of neoantigens. Alternatively, the PrP^C accumulation could be the consequence of autoimmune destruction of β -cells.