Comment on: Schuit et al. β-Cell–Specific Gene Repression: A Mechanism to Protect Against Inappropriate or Maladjusted Insulin Secretion? Diabetes 2012;61:969–975

Guy A. Rutter and Timothy J. Pullen

We write in connection to the recent review article by Schuit et al. (1) on gene repression in the β-cell. Although most review articles display a natural (and forgivable) bias toward the authors' own contributions and point of view, we feel that this article goes well beyond the norm.

Firstly, the section entitled "How islet-specifically repressed genes were identified" is largely restricted to the authors' own work. This significantly distorts the true historical narrative. It fails to mention, in particular, that the founder members (*LDHA* and *MCT1/SLC16A1*) of what were later dubbed the "disallowed" group were, in fact, first identified in a systematic comparison of the metabolic enzymology of fluorescence-activated cell sorting–purified β-cells versus a range of insulinoma cells and a limited number of primary mouse tissues (notably liver and islet non–β-cells) in a collaboration between G.A.R. and Claes Wollheim reported in 1994 (2). A further article by Matschinksy and colleagues (3), which also demonstrated low levels of lactate dehydrogenase in a relatively well-differentiated β-cell line, also goes uncited, as well as a much earlier paper by Hellman et al. (4) suggesting lactate dehydrogenase activity may be unusually low in the islet.

An additional, and greater concern, is that the review by Schuit et al. (1) fails to cite the first bioinformatics analysis to provide a list of "disallowed" genes in the β-cell (5), referring instead only to the later article from the authors' own group (6). Similarities and differences between the gene lists generated by these two articles are therefore not discussed. Strikingly, both analyses indicate that *LDHA* and *MCT1* are probably the most strongly (selectively) repressed β-cell genes (5,6), further emphasizing the importance of our original findings (2).

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From the Section of Cell Biology, Department of Medicine, Imperial College London, London, U.K.
Corresponding author: Guy A. Rutter, g.rutter@imperial.ac.uk.
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