Errata

Bennett RG, Hamel FG, Duckworth WC: Characterization of the insulin inhibition of the peptidolytic activities of the insulin-degrading enzyme–proteasome complex. *Diabetes* 46:197–203, 1997

On page 198, column 2, sentences 2, 6, and 7 under the subheading "Kinetics experiments" give the wrong units for the concentrations used in the experiments. The sentences should read as follows:

The LLVY or LSTR concentration was varied from 3 to 67 μmol/l, and the insulin concentration was set at 1.0 nmol/l, 10 nmol/l, 50 nmol/l, 0.1 μmol/l, 0.5 μmol/l, or 1.0 μmol/l.... LLVY degradation was measured as above with varying concentrations of α-casein (0, 2.1, 8.5, and 21 μmol/l). LLVY concentrations were 10, 50, and 100 μmol/l.

Please note that the units given in Fig. 6 on page 201 are correct.

Cersosimo E, Molina PE, Abumrad NN: Renal glucose production during insulin-induced hypoglycemia. *Diabetes* 46:643–646, 1997

The authors would like to correct the values calculated for right renal glucose production and utilization rates on page 644, column 2, paragraph 2, sentences 10 and 11 (lines 38–44) and on page 645, sentence 1 of the DISCUSSION section. The correct sentences appear below.

Right renal glucose production was equal to glucose utilization (0.6 ± 0.2 vs. 0.4 ± 0.2 μmol · kg^{-1} · min^{-1}) in the baseline, and net renal glucose balance was neutral (–0.2 ± 0.2 μmol · kg^{-1} · min^{-1}). Systemic hypoglycemia was associated with an increase in the rate of glucose production across the right kidney to 3.2 ± 0.2 μmol · kg^{-1} · min^{-1} ($P < 0.05$); the rate of glucose utilization increased fivefold to 1.9 ± 0.2 μmol · kg^{-1} · min^{-1} ($P < 0.01$).

The present studies confirm previous studies in dogs (11) and humans (12) indicating that postabsorptive renal glucose production is responsible for 15–20% of systemic glucose production.

Liang P, Hughes V, Fukagawa NK: Increased prevalence of mitochondrial DNA deletions in skeletal muscle of older individuals with impaired glucose tolerance: possible marker of glycemic stress. *Diabetes* 46:920–923, 1997

The authors would like to make two corrections to Fig. 1 on page 921. ND1 and ND2 (between 3 and 6 kb) are complex I genes, and COIII (between 9 and 10 kb) is part of complex IV and represents the gene for cytochrome oxidase C, subunit III. The corrected figure appears below.

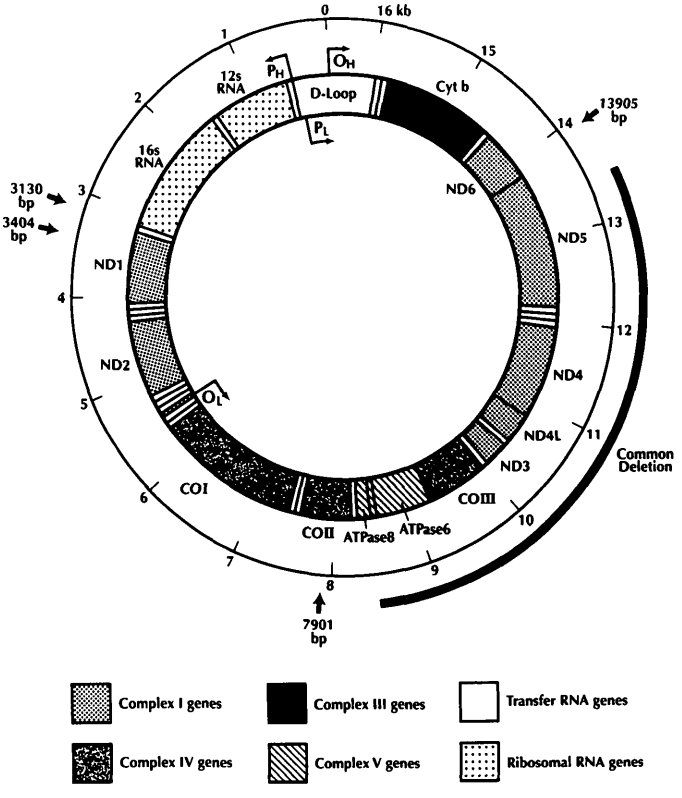

FIG. 1. Map of human mitochondrial DNA. Nucleotides 0–16569 are numbered counterclockwise from the center of the displacement (D)-loop. Shaded areas represent genes. O_H and O_L are the origins of H- and L-strand replication. P_L and P_H are the L- and H-strand promoters, respectively. The region removed by the 4,977-bp deletion is delineated by the arc outside the mtDNA circle. Arrows denote the primers selected for PCR. Oxidative phosphorylation subunits: complex I, NADH dehydrogenase (ND1, ND2, ND3, ND4); complex III, cytochrome b; complex IV, cytochrome C oxidase I, II, III; complex V, ATPase 6, ATPase 8.