Errata

Orho-Melander M, Klannemark M, Svensson MK, Ridderstråle M, Lindgren CM, Groop L: Variants in the calpain-10 gene predispose to insulin resistance and elevated free fatty acid levels. *Diabetes* 51:2658–2664, 2002

In Table 3 of the above-listed article, the authors would like to make the following corrections:

- The title of the table should be "Allele frequencies of *CAPN10* SNPs in Finland."
- For SNP-19, the insertion allele is allele 2 [i.e., "2 (ins)"] and the deletion allele is allele 1 [i.e., "1 (del)"], as indicated in the text.
- Correspondingly, the part of the legend concerning SNP-19 should read as follows: "SNP-19 allele 1 was more common among control subjects from Botnia II compared with control subjects from Botnia I (47.2 vs. 38.5%; $P = 0.041$)."

TABLE 3
Allele frequencies of *CAPN10* SNPs in Finland

		Botnia I			Botnia II			Pooled		
A. Marker	Allele	Type 2 diabetes	Control subjects	*P*	Type 2 diabetes	Control subjects	*P*	Type 2 diabetes	Control subjects	*P*
SNP-44	1 (T)	293 (78.3)	288 (75.0)	0.28	327 (80.5)	173 (81.6)	0.75	620 (79.5)	461 (77.3)	0.34
	2 (C)	81 (21.7)	96 (25.0)		79 (19.5)	39 (18.4)		160 (20.5)	135 (22.7)	
SNP-43	1 (G)	294 (76.6)	259 (67.4)	0.0049	301 (74.5)	153 (72.9)	0.66	595 (75.5)	412 (69.4)	0.011
	2 (A)	90 (23.4)	125 (32.6)		103 (25.5)	57 (27.1)		193 (24.5)	182 (30.6)	
SNP-19	2 (ins)	217 (56.5)	236 (61.5)	0.16	229 (56.4)	112 (52.8)	0.40	446 (56.5)	348 (58.4)	0.47
	1 (del)	167 (43.5)	148 (38.5)		177 (43.6)	100 (47.2)		344 (43.5)	248 (41.6)	
SNP-63	1 (C)	356 (92.7)	369 (96.6)	0.017	346 (85.2)	183 (86.3)	0.71	702 (88.9)	552 (92.9)	0.010
	2 (T)	28 (7.3)	13 (3.4)		60 (14.8)	29 (13.7)		88 (11.1)	42 (7.1)	

Data are *n* (%). All SNPs were genotyped in 395 patients with type 2 diabetes and 298 control subjects. In all, 0.3% of the genotypes could not be provided despite repeated genotyping. Allele frequencies of SNP-43 and -44 did not significantly differ between Botnia I and II samples, whereas SNP-19 allele 1 was more common among control subjects from Botnia II compared with control subjects from Botnia I (47.2 vs. 38.5%; $P = 0.041$). The SNP-63 allele 2 was substantially more common among both type 2 diabetic patients and control subjects in Botnia II than in Botnia I (14.8 vs 7.3, [$P = 0.00080$] and 13.7 vs. 3.4% [$P = 0.000013$] for type 2 diabetic patients and control subjects in Botnia II and I, respectively). All genotype frequencies were in Hardy-Weinberg equilibrium, and those of SNP-43 and -63 differed significantly between type 2 diabetic patients and healthy control subjects (SNP-43: 57.6, 35.8, and 6.6 vs. 48.2, 42.4, and 9.4%; $P = 0.039$; SNP-63: 79.8, 18.2, and 2.0 vs. 87.2, 11.5, and 1.4%, $P = 0.036$ for genotypes 11, 12, and 22, respectively).

Hanley AJG, Williams K, Gonzalez C, D'Agostino RB Jr, Wagenknecht LE, Stern M, Haffner SM: Prediction of type 2 diabetes using simple measures of insulin resistance: combined results from the San Antonio Heart Study, the Mexico City Diabetes Study, and the Insulin Resistance Atherosclerosis Study. *Diabetes* 52:463–469, 2003

In rows 13 and 19 in Table 2 of the above article, the indexes are incorrectly printed. Despite the errors in the printed formulae, all analyses presented in the article were conducted using the correct formulae, which are listed below.

Row 13 ["Belfiore (basal)"]:

$$2/[(I_0/mI_0 \times G_0/mG_0) + 1]$$

where I_0 = fasting insulin (μU/ml), mI_0 = mean fasting insulin (μU/ml), G_0 = fasting glucose (mg/dl), and mG_0 = mean fasting glucose (mg/dl).

Row 19 ("Gutt $ISI_{0,\ 120}$"):

$$(m/MPG)/\log_{10} MSI$$

where $m = [75{,}000 + (G_0 - G_2) \times 10 \times 0.19 \times W]/120$; $MPG = [(G_0 + G_2)/18]/2$; G_2 = 2-h glucose (mg/dl); W = body weight (kg); $\log_{10}$ = logarithm, base 10; $MSI = (I_0 + I_2)/2$; and I_2 = 2-h insulin (μU/ml).

The authors regret these errors and any confusion they may have caused.

Schiekofer S, Andrassy M, Chen J, Rudofsky G, Schneider J, Wendt T, Stefan N, Humpert P, Fritsche A, Stumvoll M, Schleicher E, Häring H-U, Nawroth PP, Bierhaus A: Acute hyperglycemia causes intracellular formation of CML and activation of ras, p42/44 MAPK, and nuclear factor κB in PBMCs. *Diabetes* 52:621–633, 2003

In the reference list of above-cited article, references 10–54 were incorrectly numbered as references 11–55. The corrected list is posted online at http://diabetes.diabetesjournals.org/content/vol52/issue3/.