

CORRECTIONS

Substrate specificity of sheep liver sorbitol dehydrogenase

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Volume 330 (1998), pp. 479–487

p. 479, last line of Synopsis, for “lack of” read “R”

p. 481, right-hand column, last line, for “(R)-1-phenyl-2-hydroxyethanone”, read “1-phenyl-2-hydroxyethanone”

p. 482, Table 2 caption should read “Kinetic constants for deoxy polyol substrates of sorbitol dehydrogenase (unrecorded chiral forms inactive)”

p. 483, right-hand column, line 43, for “eight distinct groups (Figure 2).” read “eight distinct groups [2] (Figure 2).”

p. 484, right-hand column, line 4, for “At C5”, read “At the non-reducing terminus”

p. 485, Figure 3 caption should read “Representation of polyol conformations (**b** is a side view of **a**)”

p. 486, reference 2 should read: Lehmann, J. (1996) in Kohlenhydrate, pp. 8–58, Georg Thieme, Stuttgart, New York

Identification of a *Caenorhabditis elegans* Δ^6 -fatty-acid-desaturase by heterologous expression in *Saccharomyces cerevisiae*

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In the above paper it was reported that the sequence of *Caenorhabditis elegans* Δ^6 -desaturase mapped to cosmid W08D2, a region of chromosome III. This chromosome assignment was incorrect, as cosmid W08D2 is derived from chromosome IV.

Actin filaments participate in the relocalization of phosphatidylinositol 3-kinase to glucose-transporter-containing compartments and in the stimulation of glucose uptake in 3T3-L1 adipocytes

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Table 4 should appear as:

Table 4 Effects of insulin and CD on GLUT4 vesicle-associated PI kinase activity

GLUT4-containing vesicles were immunopurified from LDM of untreated, insulin-stimulated (3.5 min), CD-pretreated (CD) or CD-pretreated insulin-stimulated (CD + insulin) adipocytes and used for the direct measurement of PI kinase activity on the vesicles. Non-immune murine IgG (NI-IgG) was used as control to analyse the PI kinase activity associated with the non-specific sedimentation of membranes from the LDM prepared from insulin-treated cells. PI kinase activity was measured directly on the immunopurified vesicles as described in the Materials and methods section and the legend to Figure 5. The radioactivity associated with phosphatidylinositol 3'-phosphate (PIP) spots from TLC plates like those shown in Figure 5(B) was determined by scraping off the spots from the plates and scintillation counting. The results were calculated as the means of c.p.m. detected in the PIP spots of three separate experiments. The untreated value was set at 1.0 and all other values were calculated relative to this. The PI kinase activity associated with GLUT4-containing vesicles from CD-pretreated insulin-stimulated cells were significantly different ($P < 0.01$, ANOVA) from the activity associated with GLUT4-containing vesicles from insulin-stimulated cells not pretreated with CD.

	PI kinase activity (relative to untreated)				
	Anti-GLUT-4-pelleted vesicles				NI-IgG-pelleted vesicles
Untreated	Insulin	CD	CD + insulin		
PI kinase activity	1.00 ± 0.03	2.15 ± 0.22	0.86 ± 0.081	1.25 ± 0.11	0.52 ± 0.080

The role of calmodulin-binding sites in the regulation of the *Drosophila* TRPL cation channel expressed in *Xenopus laevis* oocytes by Ca^{2+} , inositol 1,4,5-trisphosphate and GTP-binding proteins

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The dateline is missing from the above paper. It is as follows:

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