Table 1 Avidin-induced inhibition of methylmalonyl-CoA decarboxylase-catalysed Na⁺ uptake into inverted vesicles

Pretreatment of vesicles	Methylmalonyl-CoA decarboxylase activity (U per mg protein)	²² Na ⁺ uptake after incubation periods of	
		0.25 min (nmol per mg protein)	1 min (nmol per mg protein)
None	0.34	1.14	0.63
Avidin-biotin complex	0.36	1.09	0.65
Avidin	0	0.11	0.15
None, methylmalonyl-CoA omitted	0	0.13	0.14

See Fig. 1 legend for experimental conditions.

decarboxylase activity (Table 1). A parallel incubation with an avidin-biotin complex, however, did not affect either of these activities, ruling out the possibility that the avidin might act on the transport system in any way other than by a complexing of the biotin prosthetic group of the decarboxylase. The products of the decarboxylation, propionyl-CoA and bicarbonate, were unable to substitute for methylmalonyl-CoA in inducing Na⁺ accumulation to any considerable extent, thus confirming that Na⁺ is transported during the decarboxylation reaction. The function of methylmalonyl-CoA decarboxylase as a Na⁺ transport enzyme was further confirmed by reconstitution experiments. When the decarboxylase was purified by affinity chromatography on avidin-Sepharose and incorporated into phospholipid vesicles by the detergent dilution method¹⁵, using octylglucoside as the detergent, the reconstituted proteoliposomes were able to concentrate Na⁺ ions about six times over the incubation medium in response to the decarboxylation of methylmalonyl-CoA.

The mechanism of the Na⁺ transport was investigated by analysing the effect of certain ionophores. If the Na⁺ transport was electrogenic, creating a membrane potential, this could be limiting for the amount of Na⁺ accumulation, as has been shown to be the case for oxaloacetate decarboxylase⁷. The membrane potential would be discharged in the presence of valinomycin and K⁺ by the electrophoretic movement of K⁺ ions. In these conditions, about twice as much Na⁺ was accumulated in the vesicles as in the valinomycin-free controls (Table 2), thereby indicating the presence of electrogenic Na⁺ transport. The uncoupler carbonylcyanide-*p*-trifluoromethoxy phenylhydrazone also increased Na⁺ uptake within the vesicles, although less efficiently. This effect is probably also due to discharging of the membrane potential. Furthermore, the result indicates that the generation of a proton gradient is not the primary event for the Na⁺ transport. As expected, the accumulation of Na⁺ inside the vesicles was completely abolished by the presence of the Na⁺ ionophore nigericin.

Thus, our results characterize methylmalonyl-CoA decarboxylase as a new transport enzyme for Na⁺ ions. The enzyme

Table 2 Effect of ionophores on ²²Na⁺ uptake into inverted membrane vesicles

Addition	²² Na ⁺ uptake after incubation for	
	0.25 min (nmol per mg protein)	1 min (nmol per mg protein)
None	1.14	0.63
Valinomycin, 22 μM	2.22	1.24
CCFP, 20 μM	1.56	0.65
Nigericin, 22 μM	0.14	0.14
None, methylmalonyl-CoA omitted	0.13	0.14

See Fig. 1 legend for experimental conditions. CCFP, carbonylcyanide-*p*-trifluoromethoxy phenylhydrazone.

is closely related to the Na⁺ pump oxaloacetate decarboxylase⁶⁻⁸ and both enzymes constitute a new group of vectorial catalysts, converting the energy liberated by decarboxylation reactions into ion gradients. It is well known that the decarboxylation of certain 'energy-rich' carbonic acids is used as a driving force in biosynthesis, for example, in the biosynthesis of fatty acids and of phosphoenolpyruvate, where the decarboxylation of malonyl-CoA and oxaloacetate, respectively, shifts the equilibrium towards synthesis. The conversion of the energy of decarboxylation reactions into Na⁺ gradients by methylmalonyl-CoA decarboxylase and oxaloacetate decarboxylase, however, is a previously unknown biological use of decarboxylation energy, one closely related to other energy converting processes such as the formation of cation gradients by ATP hydrolysis.

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Erratum

The article by F. W. Alt, N. Rosenberg, R. J. Casanova, E. Thomas and D. Baltimore, *Nature* **296**, 325-331, was given the incorrect title. It should read 'Immunoglobulin heavy chain class switching and inducible expression in an Abelson murine leukaemia virus transformed cell line'.

Corrigenda

In the article 'Molecular cloning establishes proenkephalin as precursor of enkephalin-containing peptide' by U. Gubler *et al.*, *Nature* **295**, 206-208 (1982), in Fig. 2 the residue at position 20, shown as Asp, should be Asn, and the sequence around it Thr-Asp-Leu-Asn-Pro-Leu.

In the article 'The highly polymorphic region near the human insulin gene is composed of simple tandemly repeating sequences' by G. E. Bell *et al.*, *Nature* **295**, 31-35 (1982), the first three lines in Table 2 should read:

Sequence	Occurrences	
	Allele 1 (λHi1)	Allele 2 (λHi2)
a, ACAGGGGTGTGGGG	15 (44.1%)	24 (53.3%)
b, ACAGGGGTCTGGGG	5 (14.7%)	6 (13.3%)
c, ACAGGGGTCTGGGG	7 (20.5%)	7 (15.6%)

and the last two lines:

Arrangement of oligonucleotides

Allele 1 5' cdi aba aba dab aab aca aaa cea faa cgh ccc b 3' (34 repeats)

Allele 2 5' cdi ifa faa aba baa aaf aba afa baa aac aaa cac baa afc ccb 3' (45 repeats)