Table 1 Effect of changes in extracellular K^+ on the intracellular Na^+ and K^+ concentrations and on the specific ouabain binding of HeLa cells

Expt	$[K]_o$	$[Na]_i$ (mmol per 1 H_2O)	$[K]_i$	Specific ouabain binding as % of control
Ref. 4	0.4→0.6	24	145	159
1	0.1→0.3	70	122	136
2	0.2→0.4	87	122	96
1	0.1→0.2	111	35	83
3	0.2→0.3	97	46	55
4	0.04→?	165	50	17

removed from the membrane (by affecting the anchoring process?).

It is possible that the mechanism controlling the density of Na^+ pumps in skeletal muscle is no different from that in other cells, but that the ion concentrations achieved by any reduction of serum $[K^+]$ differ. This could be investigated further by looking at the effects of smaller reductions of serum $[K^+]$ on the ouabain binding and $(Na^+ + K^+)$ATPase of rat skeletal muscle.

Control cells had a $[Na]_i$ of 16 mmol l^{-1} and a $[K]_i$ of 179 mmol l^{-1}. The $[K]_o$ values are the initial and final values (where measured).

J. F. LAMB
J. F. AITON
PAT OGDEN

Department of Physiology and Pharmacology, Bute Medical Buildings, St Andrews, Fife KY16 9TS, UK

1. Nørgaard, A., Kjeldsen, K. & Clausen, T. *Nature* **293**, 739–741 (1981).
2. Chan, P. C. & Sanslone, W. R. *Archs Biochem. Biophys.* **134**, 48–52 (1969).
3. Erdman, E., Bolte, H.-D. & Lüderitz, B. *Archs Biochem. Biophys.* **145**, 121–125 (1971).
4. Boardman, L., Huett, M., Lamb, J. F., Newton, J. P. & Polson, J. M. *J. Physiol., Lond* **241**, 771–794 (1974).
5. Pollack, L. R., Tate, E. H. & Cook, J. S. *J. cell. Physiol.* **106**, 85–97 (1981).
6. Lamb, J. F. & Ogden, P. *Q. Jl exp. Physiol.* **67**, 105–119 (1982).
7. Baker, P. F. *Endeavour* **25**, 166 (1966).

NØRGAARD *ET AL.* REPLY—Our observation that K^+-depletion in mice and rats leads to a progressive decrease in the number of ^{3}H-ouabain binding sites in skeletal muscle[1], does not agree with the results of previous investigators, who found that K^+ depletion increases $(Na^+ + K^+)$ATPase and the number of ^{3}H-ouabain binding sites in erythrocytes[2,3] as well as $(Na^+ + K^+)$ATPase activity in guinea pig hearts[4,5]. Our observation that erythrocytes of K^+-depleted rats maintain normal K^+-contents even after weeks of exposure to the low plasma K^+ values agrees with the existence of a compensatory rise in their number of $(Na^+ + K^+)$ATPase units. The reported measurements[4,5] of $(Na^+ + K^+)$ATPase activity in hearts used a sediment obtained by centrifugation of homogenates, and as this may contain only a minor fraction of the total $(Na^+ + K^+)$ATPase activity present in the starting material (for discussion, see ref. 6), the results depend on the recovery being the same for hearts from normal and K^+-depleted animals.

To explore this problem further, we have recently measured the binding of ^{3}H-ouabain to rat heart ventricles *in vivo*. In the controls we found 276 ± 8 pmol per g wet wt, and 157 ± 18 pmol per g in the K^+-depleted rats ($P < 0.001$). This decrease (43%) is smaller than that found in skeletal muscle, which agrees with the observation that the loss of K^+ was not so pronounced in the heart. Measurements of the ^{3}H activity in the plasma after intraperitoneal injection of ^{3}H-ouabain gave 77% higher values in the K^+-depleted rats than in the controls. This also suggests that the peripheral binding capacity for ^{3}H-ouabain is reduced and that in the K^+-depleted state the heart may be exposed to a higher concentration of digitalis glycosides.

At variance with the results obtained using HeLa cells[7], even modest K^+ loss (and a corresponding rise in intracellular Na^+) is associated with a decrease in the number of ^{3}H-ouabain binding sites in rat skeletal muscle (see Figs 1, 2 of ref. 1). This suggests that the synthesis of new $(Na^+ + K^+)$ATPase units has ceased, and we are now investigating the possibility that this results from a general impairment of enzyme activity or protein synthesis.

Despite our original expectations, we have been unable to detect any rise in the number of ^{3}H-ouabain binding sites in the muscles of K^+-depleted mice or rats. It seems that skeletal and heart muscle cells differ from cultured cells, perhaps due to central regulatory mechanisms triggered by the drop in plasma K^+ seen a few days after the onset of the K^+-depleting regime. The decreased capacity for active $Na^+ + K^+$ transport in muscle may prevent any further reduction in plasma K^+ and delay the development of the muscle paralysis which is the final outcome of prolonged K^+ depletion.

AAGE NØRGAARD
KELD KJELDSEN
TORBEN CLAUSEN

Institute of Physiology, Aarhus University, DK-8000 Aarhus C, Denmark

1. Nørgaard, Aa., Kjeldsen, K. & Clausen, T. *Nature* **293**, 739–741 (1981).
2. Chan, P. C. & Sanslone, W. R. *Archs Biochem. Biophys.* **134**, 48–52 (1969).
3. Erdmann, E. & Krawietz, W. *Acta biol. med. germ.* **36**, 879–883 (1977).
4. Erdmann, E., Bolte, H.-D. & Lüderitz, B. *Archs Biochem. Biophys.* **145**, 121–125 (1971).
5. Bluschke, V., Bonn, R. & Greeff, K. *Eur. J. Pharmac.* **37**, 189–195 (1976).
6. Clausen, T., Hansen, O. & Larsson, L.-I. *Eur. J. Pharmac.* **72**, 331–335 (1981).
7. Boardman, L., Huett, M. & Lamb, J. F. *J. Physiol., Lond.* **241**, 771–794 (1974).

The active radio galaxy 1413+135

BEICHMAN *et al.*[1] and Bregman *et al.*[2] present interesting IR and high-frequency radio (4.8–14.5 GHz) results on 1413+135. The latter also give an optical spectrum which suggests that it is a galaxy of redshift $z = 0.26$; no emission features were seen. Curiously Beichman *et al.*[1] imply that it was an 'empty field' until discovered as an IR source by Rieke *et al.*[3].

The radio source was identified by Hoskins *et al.*[4] with a 20 mag galaxy on the Palomar Sky Survey, and a finding chart was given. The identification was based on 408-MHz measurements at Molonglo. Condon *et al.*[5] obtained an accurate radio position which agrees to 1 arc s with the optical position of Hoskins *et al.*[4]. This confirmed the identification but as Bregman *et al.*[2] point out, the finding chart of Condon *et al.*[5] is faulty. Their positional offsets are also misleading. Bregman *et al.*[2] reconfirm the identification of Hoskins *et al.*[4] but mistakenly refer to this as a 'PKS identification'.

A radio spectrum covering the range 178–2,695 MHz is given by Murdoch and Hoskins[6]. The low-frequency spectrum peaks at 3 Jy at ~300 MHz falling to 1.9 Jy at 178 MHz and 0.67 Jy at 2,695 MHz (in 1971). Bregman *et al.*[2] report a strong and variable rise at 4.8 GHz and above and Beichman *et al.*[2] show that the intensity peaks at ~100 GHz. In addition to the highly compact component implied by these results, there is evidently a moderately compact component shown by the low-frequency radio data. The overall radio spectrum is quite typical of BL Lac objects and highly variable QSOs. The object would appear to be a galaxy with a BL Lac object in its nucleus.

H. S. MURDOCH

The Astrophysical Department, School of Physics, University of Sydney, New South Wales 2006, Australia

1. Beichman, C. A. *et al. Nature* **293**, 711 (1981).
2. Bregman, J. N. *et al. Nature* **293**, 714 (1981).
3. Rieke, G. H., Lebofsky, M. J. & Kinman, T. D. *Astrophys. J. Lett.* **232**, L151 (1979).
4. Hoskins, D. G., Murdoch, H. S., Hazard, C. & Jauncey, D. L. *Aust. J. Phys.* **25**, 559 (1972).
5. Condon, J. J., Hicks, P. D. & Jauncey, D. L. *Astr. J.* **82**, 692 (1977).
6. Murdoch, H. S. & Hoskins, D. G. *Aust. J. Phys.* **26**, 867 (1973).

0028-0836/82/080718—01$01.00