

**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	$[K^+]_o$ (mmol per 1 $H_2O$ )	$[Na^+]_i$	$[K^+]_i$	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  $16 \text{ mmol l}^{-1}$  and a  $[K^+]_i$  of  $179 \text{ mmol l}^{-1}$ . The  $[K^+]_o$  values are the initial and final values (where measured).

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- Nørgaard, A., Kjeldsen, K. & Clausen, T. *Nature* **293**, 739–741 (1981).
- Chan, P. C. & Sanslone, W. R. *Archs Biochem. Biophys.* **134**, 48–52 (1969).
- Erdmann, E., Bolte, H.-D. & Lüderitz, B. *Archs Biochem. Biophys.* **145**, 121–125 (1971).
- Boardman, L., Huett, M., Lamb, J. F., Newton, J. P. & Polson, J. M. *J. Physiol., Lond.* **241**, 771–794 (1974).
- Pollack, L. R., Tate, E. H. & Cook, J. S. *J. cell. Physiol.* **106**, 85–97 (1981).
- Lamb, J. F. & Ogden, P. Q. *Jl exp. Physiol.* **67**, 105–119 (1982).
- 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  $^3H$ -ouabain binding sites in skeletal muscle<sup>1</sup>, does not agree with the results of previous investigators, who found that  $K^+$  depletion increases  $(Na^+ + K^+)ATPase$  and the number of  $^3H$ -ouabain binding sites in erythrocytes<sup>2,3</sup> as well as  $(Na^+ + K^+)ATPase$  activity in guinea pig hearts<sup>4,5</sup>. 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<sup>4,5</sup> 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  $^3H$ -ouabain to rat heart ventricles *in vivo*. In the controls we found  $276 \pm 8 \text{ pmol per g wet wt}$ , and  $157 \pm 18 \text{ pmol per g}$  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  $^3H$  activity in the plasma after intraperitoneal injection of  $^3H$ -ouabain gave 77% higher values in the  $K^+$ -depleted rats than in the controls. This also suggests that the peripheral binding capacity for  $^3H$ -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<sup>7</sup>, even modest  $K^+$  loss (and a corresponding rise in intracellular  $Na^+$ ) is associated with a decrease in the number of  $^3H$ -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  $^3H$ -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.

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- Nørgaard, A., Kjeldsen, K. & Clausen, T. *Nature* **293**, 739–741 (1981).
- Chan, P. C. & Sanslone, W. R. *Archs Biochem. Biophys.* **134**, 48–52 (1969).
- Erdmann, E. & Krawietz, W. *Acta biol. med. germ.* **36**, 879–883 (1977).

- Erdmann, E., Bolte, H.-D. & Lüderitz, B. *Archs Biochem. Biophys.* **145**, 121–125 (1971).
- Bluschke, V., Bonn, R. & Greeff, K. *Eur. J. Pharmac.* **37**, 189–195 (1976).
- Clausen, T., Hansen, O. & Larsson, L.-I. *Eur. J. Pharmac.* **72**, 331–335 (1981).
- Boardman, L., Huett, M. & Lamb, J. F. *J. Physiol., Lond.* **241**, 771–794 (1974).

## The active radio galaxy 1413+135

BEICHMAN *et al.*<sup>1</sup> and Bregman *et al.*<sup>2</sup> 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.*<sup>1</sup> imply that it was an 'empty field' until discovered as an IR source by Rieke *et al.*<sup>3</sup>.

The radio source was identified by Hoskins *et al.*<sup>4</sup> 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.*<sup>5</sup> obtained an accurate radio position which agrees to 1 arc s with the optical position of Hoskins *et al.*<sup>4</sup>. This confirmed the identification but as Bregman *et al.*<sup>2</sup> point out, the finding chart of Condon *et al.*<sup>5</sup> is faulty. Their positional offsets are also misleading. Bregman *et al.*<sup>2</sup> reconfirm the identification of Hoskins *et al.*<sup>4</sup> 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<sup>6</sup>. 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.*<sup>2</sup> report a strong and variable rise at 4.8 GHz and above and Beichman *et al.*<sup>2</sup> 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.

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