Uremia and Red Blood Cell Lithium-Sodium Countertransport

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Dear Sir,

Recently Diez et al. [1] have addressed the issue of the relationship between uremia and red blood cell sodium transport. Among the different erythrocyte sodium transport systems studied, the authors report on the lithium-stimulated sodium efflux and conclude that this pathway is not altered in uremic patients. We believe that the interpretation of the data by Diez et al. is incorrect. Hemodialysis patients show on the average an increase of about 24% compared to both controls and transplant recipients (105 vs. 85 $\mu\text{mol/L}$ RBC/h), and, contrary to what the authors state, this difference does reach statistical significance. Using the unpaired t test formula on the authors' data, a t of 2.45 is calculated, with 39 degrees of freedom (p value < 0.02). Analysis of variance with multiple comparison procedure using the Scheffe method gives similar results. Transplant recipients have average values of lithium-stimulated sodium efflux identical to controls. The difference between transplant recipients and hemodialysis patients does not reach statistical significance because of the limited sample size involved in the comparison, resulting in a reduced statistical power. In a forthcoming paper [2], we have compared the lithium-stimulated sodium efflux between male hemodialysis patients and a group of controls. Patients, on the average, have a 28% increase in the maximal velocity of the lithium-stimulated sodium efflux compared to controls and this difference reaches statistical significance at the 0.05 level. Both the findings of Diez et al. [1] and our own findings are in agreement with the suggestion of Woods et al. [3] of an increased countertransport in uremic patients. The conclusions therefore from the report of Diez et al. [1] should be that, in addition to the ouabain-sensitive Na efflux (Na-K pump) and the bu-metamide-sensitive Na efflux (Na/K cotransport), uremia affects the lithium-stimulated sodium efflux (Na-Li countertransport) and that this latter alteration seems to be reversed after renal transplantation. The difference detected between the hemodialysis patients and controls in the report of Diez et al. is similar to the difference found between male normotensives and essential hypertensive patients [4]. The possibility that hypertensive and uremic patients share similar abnormalities in the lithium-stimulated lithium efflux is interesting and needs to be explored further. The study of ion transport in uremic patients could help, in fact, to gain important insights into the pathophysiology on both hypertension and uremia.

References

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