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LETTER TO THE EDITOR

Hydration increases cell metabolism

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The recent article by Mathai *et al.*¹ published in the *International Journal of Obesity* entitled 'Selective reduction in body fat mass and plasma leptin induced by angiotensin-converting enzyme inhibition in rats' is very interesting for a number of reasons but perhaps one of the most important is the fact that the treated rats drank twice the amount of water (about 80 ml day⁻¹) as the non-treated controls (about 40 ml day⁻¹). This is a large difference that would have the effect of increasing the flow of water through the body. The authors discuss this point and cite four other studies that have also found increased water intake with renin-angiotensin system inhibition. They suggest possible mechanisms for this enhanced drinking as being due to an increased presence, and thus action, of angiotensin in the brain due to ACE inhibition in the periphery and/or due to urine loss. Both of these are credible physiological mechanisms but they need to be demonstrated.

Parallel to this increased water intake, in this study and the four other studies cited (plus other studies referenced but where water intake was not measured), inhibition of the renin-angiotensin system produced a marked decrease in adiposity. This could suggest that either inhibition of the renin-angiotensin system unblocks fat metabolizing mechanisms, or that the increased water intake *per se* allows cells to hydrate better and thus regulate fat metabolism better.

It has been noted that inhibition of the renin-angiotensin system allows cells to restore glucose transport across the cell membrane as well as to increase their sensitivity to insulin.² This effect would also restore normal cellular carbohydrate metabolism. What is observed though is an enhanced fat metabolism, which in the long run might be underlying the increased insulin sensitivity.

An increased water intake would decrease plasma osmolality, which would decrease vasopressin levels in the blood, which in turn would allow abundant urine excretion. An animal model of this condition is the Brattleboro rat in which the homozygous di/di condition does not produce any vasopressin. These animals can drink and excrete up to 250 ml day⁻¹. Their growth is slower than the controls, they are lean and their plasma levels of leptin are reduced,³ the latter findings being similar to the Mathai study. Furthermore, neuronal metabolism of glucose is enhanced,

suggesting that cells are functioning normally under appropriate physiological conditions.⁴

All these changes would suggest an improved metabolic function, in particular improved lipid metabolism in animals drinking increased amounts of water. It has been shown that cell dehydration inactivates mTOR (mammalian target of rapamycin) signalling and decreases insulin-induced glucose uptake.⁵ Thus, cell hydration should have the opposite effect and enhance insulin-induced glucose uptake as well as normal metabolic function.

It would be interesting to propose that increased water intake due to renin-angiotensin system blockade be investigated more systematically in studies on obesity and diabetes, as suggested by the results of Bilz *et al.*⁶ and Keller *et al.*⁷ from human studies where they showed that transient hypo-osmolality increased whole-body lipid turnover.

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