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Lithium in drinking water and food, and risk of suicide

Ohgami *et al*[1] reported lithium in drinking water (0.7–59 gm/l) and linked it to suicide rates. However, dietary lithium, which has received scant attention, is found in grains and vegetables, and to some extent animal-derived foods.[2] Hence, considering only lithium in drinking water may not be enough of a link to suicide rates. Dietary sources of lithium may actually have made the difference rather than just the drinking water. Differences in the prevalence of mood disorders with natural lithium levels acting as a prophylactic have been reported.[3,4] Jathar *et al*[3] assessed the lithium content of the daily diet (72.55–154.6 µg) and biological fluids, and hypothesised lithium to be a natural prophylactic. It will be interesting to see whether dietary and drinking water lithium levels have a direct impact on mood disorder prevalence, which in turn could explain the variation in suicide rates. And what about lithium-containing food cooked in lithium-containing tap water?

1 Ohgami H, Terao T, Shiotsuki I, Ishii N, Iwata N. Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 464–5.

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The study by Ohgami *et al*[1] raises serious ethical issues related to the interpretation of research findings and, as a consequence, their possible application. While not denying that the findings are interesting and have caused a stir in the lay press and on the internet, we question the methodology and the possible implications if the results are taken seriously.

First, sociological reasons for suicide are important, and changing rates of suicide in many countries are linked to changes such as migration, poverty, relationships and economic issues. The finding that when gender was included in the analysis there was a difference in the significance levels between men and women (with the results being less significant in women) is one such example. Adding lithium to tap water is not going to change these demographic and social factors that contribute to suicide rates, and not having accounted for at least some of these is a major limitation of the study. Second, although we agree with Young[2] in his commentary that more research is needed to prove or disprove this tantalising idea, it is also important to assess what the impact of different levels of tap-water lithium is going to be on thyroid function, pregnant women and on the unborn fetus. It is also important to assess whether tap-water levels of lithium directly correlate with serum lithium levels in the respective populations. The levels of lithium in body fluids in normal healthy controls have varied from 0.01 to 0.09 meq/1 in one study,[3] but there are no data about serum lithium levels among individuals attempting suicide. Maybe assessment of serum lithium levels among those with suicidal behaviour can be a place to start. More data are also needed on the role of low-dose lithium in individuals without mood disorders who are at risk of suicide.

Finally, several foods (particularly spices) are known to have relatively high levels of lithium as reported by a study in India several years ago.[3] This study reported levels as high as 12 µg/g of lithium in tobacco and high levels in crude salt, rock salt and several spices. Maybe, until such time that we are certain about lithium's role in decreasing suicidality in non-psychiatric populations, it might be worth conducting randomised controlled trials with these foods in individuals with suicidal behaviour to see whether low doses of lithium really help.

Let us not throw the lithium out with the tap water yet!

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Authors' reply: We thank Drs Chandra and Babu for their comments, but we would like to emphasise that we had never recommended the addition of lithium to drinking water supplies[1] because our findings are preliminary and yet to be conclusive.

First, we agree that sociological factors such as migration, poverty, human relations and economic issues may be associated with suicide rates, and have already admitted such limitations by stating 'other factors such as psychosocial and economic factors were not taken into consideration'.[1] Second, Drs Chandra and Babu state that it is also important to assess side-effects of lithium in tap water on thyroid function, pregnant women and the unborn fetus. Although it seems probable that these low levels of lithium are far below the levels required to produce side-effects, we agree with them. Third, they mention lithium levels in food, also raised by Drs Desai and Chaturvedi. This may be important because dietary lithium intake is estimated not to be a negligible quantity. For example, mean (s.d.) dietary lithium was reported to be: 1560 µg/day (980) in China; 1485 (1009) (Tijuana) and 939 (928) (Culiacan) in Mexico; 1090 (324) in Sweden; 1009 (324) in Denmark; 821 (684) (Texas), 650 (740) (New York) and 429 (116) (San Diego) in the USA; 812 (383) in Japan; 406 (383) in Germany; and 348 (290) in Austria.[2] Therefore, at the next stage,

it would seem necessary to measure serum lithium levels in participants, incorporating total lithium intake of both drinking water and food.

1 Ohgami H, Terao T, Shiotsuki I, Ishii N, Iwata N. Lithium levels in drinking water and risk of suicide. *Br J Psychiatry* 2009; **194**: 464–5.

2 Schrauzer GN. Lithium: occurrence, dietary intakes, nutritional essentiality. *J Am Coll Nutr* 2002; **21**: 14–21.

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Psychosis and catatonia as a first presentation of antiphospholipid syndrome

We report (with the patient's consent) a 28-year-old woman who presented with episodic psychosis and catatonia associated with antiphospholipid syndrome, with venous thromboembolism, rash, an acute phase response, and elevated liver enzymes. We know of no previous reports of catatonia associated with this syndrome.

She was admitted abroad in October 2007 with rapid-onset psychosis (persecutory delusions, visual/auditory hallucinations), confusion, and disorientation. She responded to quetiapine and lorazepam, and initially remained well after stopping medication. In July 2008 she deteriorated, with low mood, somatic and nihilistic delusions, and demotivation. She was admitted with catatonic stupor, staring and mutism. She improved with haloperidol, exhibiting severe distraction with thought block and hypersensitivity to background noise, before recalling visual/auditory hallucinations, confusion and delusions. In August 2008 she suffered a spontaneous popliteal vein thrombosis and a mild purpuric rash.

She had no personal or family history of psychiatric, autoimmune or thromboembolic disease, did not smoke or use recreational drugs, and took no medication except an oral contraceptive pill briefly before, and olanzapine the day before, admission (July 2008).

She had persistent elevations in alanine aminotransferase (79 U/l prior to quetiapine, peak 257 U/l), erythrocyte sedimentation rate (19–24 mm/h), and C-reactive protein (17 mg/l). Hepatic ultrasound showed mild diffuse echogenicity. Anticardiolipin antibodies were positive (22 IgMU/ml, August 2008; 25.4 IgGU/ml, October 2008; 18.0 IgMU/ml, November 2008 after immunosuppression). Antinuclear antibody was negative from October 2007 to August 2008, but weakly positive in October 2008. Rheumatoid factor likewise became positive.

Normal investigations included head magnetic resonance imaging, electroencephalography, blood count, renal/thyroid function, electrolytes, calcium/phosphate, folate, cobalamin, ceruloplasmin, ammonia, lactate, porphyrins, amino/organic acids, complement, lupus anticoagulant, serology for hepatitis A/B/C, cytomegalovirus, Epstein–Barr virus, syphilis, *Toxoplasma*, and HIV; and antimitochondrial, anti-smooth muscle, anti-liver–kidney microsome, anti-thyroid peroxidase, anti-Hu/Ri/Yo, anti-voltage-gated potassium channel, anti-*N*-methyl-*D*-aspartate receptor, anti-myeloperoxidase, anti-proteinase-3, and anti-β_2-glycoprotein-1 antibodies. Hepatic ultrasound showed mild diffuse echogenicity.

Following anticoagulation, haloperidol and venlafaxine, she was anticoagulated further (international normalised ratio 3:4) and immunosuppressed (azathioprine, prednisolone), leading to symptomatic resolution.

Vascular thrombosis and persistent antiphospholipid antibodies constitute antiphospholipid syndrome.[1] Catatonic immobility may have contributed to her thrombosis, but does not explain the immunophenotype. Oral contraceptives can exacerbate antiphospholipid syndrome; oral contraceptive use and antiphospholipid antibodies may be associated, but primarily for anti-β_2-glycoprotein-1 antibodies.[2] Phenothiazines can induce antiphospholipid antibodies, but this has not been reported after quetiapine, olanzapine, or haloperidol. Although our patient may represent the first such occurrence, the spontaneous inflammation suggests an alternative interpretation. Research criteria for systemic lupus erythematosus were not met, but her inflammatory disorder may be an early stage of this disease. Psychosis and catatonia can occur in lupus. Antiphospholipid antibodies are associated with neuropsychiatric manifestations of systemic lupus erythematosus[3] and psychosis *per se*.[4]

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