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Psychiatric morbidity in the relatives of schizophrenic probands

SIR: We were puzzled by some of the figures presented by Varma & Sharma (*Journal*, May 1993, **162,** 672–678) in their paper. In the methods section, having started with the first-degree relatives of 162 patients and 106 controls (a maximum of 324 and 212 parents respectively), 11 parents of the schizophrenic group and four parents of the control group were excluded because of inadequate data. Despite these exclusions, data presented on the smaller samples still included 324 parents of schizophrenics and 212 parents of controls, the maximum possible number. We would be grateful for clarification from the authors as this would help us to evaluate the potentially interesting results presented on this large dataset.

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AUTHOR'S REPLY: In response to Walsh & Gill I would like to clarify that we had deliberately excluded 11 parents of the schizophrenic group and four parents of the control group for uniformity sake. This was done because they were step-parents and their number was too small for any significant analysis. In our study biological parents were taken into consideration. This is the reason for the 324 and 212 parents being present in our analysis.

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Lithium toxicity at therapeutic serum levels

SIR: Bell *et al* (*Journal*, May 1993, **162,** 689–692) have suggested monitoring of red blood cell (RBC) and plasma lithium levels in patients who develop lithium toxicity. However, they have neither given the RBC lithium levels nor demonstrated the necessity for monitoring them concurrently with the serum levels. The following case elucidates the importance of this investigation.

Case report. S, a 45-year-old woman with recurrent bipolar affective disorder of eight years' duration, has been on prophylactic lithium (900 mg/day with a serum lithium level of 1.05 mmol/l at the last follow-up three months ago) for five years. She was admitted with a history of withdrawal (eight days), occasional vomiting (four days) and stupor (24 hours). On examination, her vital parameters were normal and she was not dehydrated. She was in altered sensorium, not responding to verbal commands and incontinent. Her metabolic parameters including serum electrolytes were normal. An x-ray of the skull, a cranial computerised tomography scan, and lumbar cerebrospinal fluid studies were also normal. Her serum lithium level at admission was 0.5 mmol/l, 12 hours after the last lithium dose (450 mg). Lithium was discontinued on admission. The serum lithium level as well as the clinical status were the same the next day.

Her RBC lithium level was 1.0 mmol/l against a corresponding plasma level of 0.5 mmol/l. Two days later the patient's sensorium had improved. She was no longer incontinent, obeyed simple commands, and indicated her needs verbally. Her RBC lithium level was 0.8 mmol/l against a plasma level of 0.2 mmol/l. Twelve days after stopping lithium the patient was completely orientated and was ambulant with no neurological deficits. Her RBC lithium level was 0.3 mmol/l and her plasma level was below 0.2 mmol/l. She was discharged one week later with no drugs. She has remained symptom-free for the past 15 months of follow-up.

For this patient, the serum lithium level alone was misleading – being at the lower end of therapeutic range and hence not toxic. However, monitoring RBC lithium levels revealed high intracellular concentrations. After stopping lithium the plasma levels decreased rapidly, whereas the RBC lithium levels showed a slower fall. The clinical status of this patient improved with the fall in RBC lithium levels. This case highlights the importance of RBC lithium level monitoring in addition to serum level and strengthens the case for the suggestion of Bell *et al*.

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