Different results were found however, when comparing lithium levels in mouse brain and in plasma (Herteaux *et al*, 1991). Lithium had a half-life ($T_{1/2}$) of between 12 and 24 hours (up to 40 hours in some cases).

Plasma lithium peaks in one to two hours (taking more for the 'slow release' preparations). The assumption that brain lithium levels will continue to rise higher than the serum lithium concentration is not conceivable.

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AUTHORS' REPLY: We cannot agree with the assertion that it is "not conceivable" for brain levels of lithium to rise higher than those of serum. Several animal studies have shown that brain lithium levels can exceed serum levels: Plenge (1978) showed brain lithium levels above serum levels eight hours after an intraperitoneal injection of lithium chloride in chronically treated rats; and Schou (1958) showed that, following intravenous administration, the brain lithium level rises slowly, reaching a maximum in 22-26 hours (i.e. when serum levels are falling). The brain lithium level remains higher than the serum lithium level, decreasing in a parallel fashion.

In one of the references cited by Dr Moscovich (Herteaux *et al*, 1991; see above) it was shown, using stable isotopes of lithium, that, while the equilibration of lithium between serum and brain was almost instantaneous, lithium levels in grey matter were three to six times higher than in plasma under steady-state conditions. Moreover, it is clear that the uptake of lithium into the brain is not uniform. Herteaux *et al* (1991) showed high lithium levels in thalamus, neocortex and hippocampus (six times greater than plasma levels), with lower levels in striatum and cerebellum (three times greater than plasma levels), and reviewed several other studies which supported this observation. This phenomenon, which has also been seen in human studies, may well be relevant to the development of lithium neurotoxicity.

Kluge *et al* (1978) showed much higher levels of lithium in the brain than found in the serum in two cases who died of lithium intoxication – the highest concentrations were in the neocortex and brain stem. Kinetic experiments suggest that lithium in the brain may be in a 'deep compartment' with which equilibration is slow. This means that central effects may develop even in the presence of falling or low serum levels, and this observation may explain why the clinical signs of lithium toxicity may persist several days after successful haemodialysis which has lead to minimal concentrations of lithium in the serum. Amdisen *et al* (1974) showed high levels of lithium in certain areas of the brain (notably in the neocortical white and grey matter) in a case of lithium toxicity who died 20 days after serum lithium had been re-stabilised at 0.4 mmol/l.

We take issue with the respondent's assertion that we 'assumed' that lithium levels in the brain continue to rise higher than serum levels in all cases, all the time. We are arguing that, under certain circumstances, lithium levels in the brain can be higher than in the serum, that key nuclei can be particularly affected, and that the central effects of lithium may persist for some time after serum levels are lowered. Inter-individual variation in serum and red blood cell ratios are probably under genetic control and it may be that those rare individuals who manifest neurotoxic symptoms at therapeutic lithium serum levels are particularly prone to have higher lithium levels in the brain (perhaps site-specific) due to a state and/or trait tendency to accumulate intracellular lithium. Indeed, a number of separate studies have shown that it is possible to predict lithium toxicity by closely monitoring the red blood cell:serum lithium ratio. There is an increase in this ratio just before a neurotoxic episode (Tyrer & Shopsin, 1980).

We agree that the evidence regarding lithium distribution is complex and at times conflicting. It would be interesting to study lithium handling in those individuals who have manifested neurotoxicity at therapeutic lithium levels to see if this is abnormal.

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Court diversion

SIR: Joseph & Potter (*Journal*, March 1993, **162**, 325–334) are to be congratulated for their work on court diversion. Their studies had clear objectives, sound methodology, and provided lucid discussion of the findings. Two of their principal results, notably the difference in the rates of custodial remands depending on which psychiatrist took responsibility for the case, and that the admissions via custody had a better outcome, were surprising.

The authors' tacit assumption of an apparent causal relationship between the drop in the combined psychiatric in-patient population between 1962 and 1990, and the rise in the combined prison population in the same period reopens an old debate on a complex issue (Penrose, 1939). Although most studies, supported by anecdotal and clinical impressions, sustain the notion that there is a discernible increase in the number of mentally ill in prison populations in recent years, and would tend to confirm Penrose's hypothesis, whether or not there is a direct effect of de-institutionalisation remains contentious. Differential arrest rates (Teplin, 1985) and changing mental health policies (Whitmer, 1990) may contribute to this increase.

The absence of any discussion on the ethical issues of obtaining informed consent and possible interference in the due process of law was disappointing (Carson, 1992). The identification of those included in the study population depended on court officials acting as 'filters', yet the discussion failed to highlight potential difficulties such 'filters' have, both in detection rates (i.e. what proportion of the target population had been already diverted, such as by use of Section 136 of the Mental Health Act (MHA) 1983 for instance?), and in their ability to discriminate (were the quiet, withdrawn individuals missed because they were not so obviously 'strange in manner'? Was there a bias towards not referring those on more serious charges?).

The authors emphasise that in order to secure admission to hospital, either informally or under the civil provisions of the MHA 1983, it was necessary to persuade the Crown Prosecution Service (CPS) to discontinue proceedings. It is certainly inaccurate to suggest that if the CPS will not discontinue proceedings, there is no possibility of either formal or informal admissions to hospital under the civil provisions. Furthermore, the criminal provisions of the MHA 1983 allow for admissions for assessment (Section 35) from the magistrates' court without a need for the CPS to discontinue proceedings. Thirty-four per cent of the psychotic group were considered unfit to plead. This is a high figure and we would appreciate further information on the criteria used.

The interesting finding that the psychiatric recommendations differed significantly depending on which psychiatrist was involved, especially if considered in the light of the better outcome in the group admitted via custody, seems to make the point that what appears best in principle (i.e. early diversion) is not necessarily borne out in practice. It is impossible to ignore that the important factor determining a better outcome was the time spent in custody before admission to hospital.

Although we were not surprised that 31% of those assessed had a lifetime history of drug abuse, we were surprised that those with a history of illicit drug abuse did not have significantly more convictions than the rest of the sample.

We feel that the resource implications outlined in their second paper are inconclusive. Various opportunity costs such as 'wear and tear' on staff and on other patients were notable by their absence. If one uses the findings to inform debate on an options appraisal for determining the service that maximises utility, there is a suggestion that one should concentrate more on the prison medical service and further explore the use of Sections 35, 36 and 48 of the MHA 1983. Greater emphasis could be placed on helping prisons improve their methods of tracking and identifying the mentally disordered as a prelude to planning treatment strategies (Jemelka *et al*, 1989).

The alarming discovery that, whereas 70% of those assessed had previously been in-patients (of whom over three-quarters had been detained under the provision of the MHA 1983), only one-quarter of those with previous admissions were receiving psychiatric support at the time of the study, makes one wonder whether it is appropriate to detect those in 'need' if efforts at follow-up are so poor. Finally, the stark reality that 22 of the 51 direct admissions from court, admitted because they were psychotic were later discharged because they continued to be psychotic is a sobering reminder of

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