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Khat-Induced Paranoid Psychosis

SIR: Gough & Cookson (*Journal*, June 1987, **150**, 875–876) mention that khat had caused psychosis in their patient as the urine screen was positive for amphetamines. It is evident that the authors use the word "amphetamines" to refer to phenylalkymines, as otherwise they would have been more specific. This implies that the test was non-specific and could have been positive for many other compounds which are in common use, for example as cough remedies. It is true that the two important active chemical constituents in khat, cathinone and d-norpseudoephedrine (DNE), which are responsible for the euphoriant properties, are both phenylalkylamines.

Our contention is that it would not be possible to differentiate cathinone and DNE from other amphetamine-like compounds in the routine urine screen for amphetamines, but a more definitive testing using chromatographic techniques would yield conclusive results. Cathinone is easily hydroxylated to form l-norpseudoephedrine and DNE in the body. The relative proportions of cathinone and norpseudoephedrine in urine may indicate the time interval between khat chewing and their excretion in the urine. Khat users would, as a matter of habit, continue to chew it, and a follow-up of the patient may yield conclusive results.

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SIR: We never suggested that "khat had caused psychosis" in our patient because the urine drug screen was positive for amphetamines, merely that this result was to be expected in view of the chemical constituents in khat. We would refer the authors to the original report (Gough & Cookson, 1984), in which the case was discussed in detail and the type of assay used specified. The patient was observed at home prior to admission actually chewing the leaves, which were identified as khat by a Regional Drug Information Centre.

Our previous letter was prompted by Critchlow & Seifert's case report (*Journal*, February 1987, **150**, 247–249), where a urine drug screen was positive for morphine and dihydrocodeine but negative for amphetamines, which is not in keeping with a diagnosis of khat-induced psychosis.

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Psychiatry and Glasnost

SIR: At the recent Regional (European) Conference of the World Association for Social Psychiatry (WASP) held in Budapest, Hungary (13–15 November), Professor Modest Kabanov of Leningrad highlighted changes in attitude stemming in part from Glasnost.

The concerns about Soviet psychiatry and allegations of its misuse are now taken seriously by Soviet academics and the Government, leading to open discussion in the press and other media. One such example was a detailed article in *Izvestia*, "In defence of the unprotected mentally ill", on 10 July, 1987. This stimulated the open-minded investigation and review of cases by independent committees, one of which was headed by Professor Kabanov. This appears to be a new development and not dissimilar to the mental health tribunal mechanism prevailing in England.

Professor Kabanov went on to describe new legislation procedures and guidelines currently being considered and soon to be implemented which will mitigate against incorrect diagnosis and the inappropriate use of psychiatry. Incorporated in this is the concept of independent assessment, including a possible contribution from foreign academics. In Leningrad these changes have already been effected, helped by the initiative of Professor Kabanov. Hopefully they will become accepted and generally implemented throughout the USSR.

Professor Kabanov concluded by stating that Soviet psychiatrists would welcome constructive discussion, and suggested a forum at the next World Conference of WASP to be convened in London in November 1988, where a key topic might be 'Global issues in social psychiatry', with active participation by Eastern Bloc socialist countries.

Finally, he put in a plea, in the words of Voltaire: "I may disapprove of what you say, but I defend to

the death your right to say it" – perhaps another expression of Glasnost.

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Urinary Tribulin Output in Patients with Panic Attacks

SIR: Tribulin is an endogenous compound present in urine, which has the properties of inhibiting monoamine oxidase (MAO) activity and displacing ^{3}H-flunitrazepam binding to brain membrane preparations (Elsworth *et al*, 1986). It has been demonstrated that urinary tribulin output is increased in disorders associated with increased anxiety: alcohol withdrawal (Bhattacharya *et al*, 1982) and benzodiazepine withdrawal (Petursson *et al*, 1982). From these and other studies it was suggested that tribulin may be an endogenous anxiety-promoting agent (Sandler, 1982). If this is the case, then in patients with anxiety disorders, such as panic disorder or agoraphobia with panic attacks, tribulin output would be expected to be elevated compared with normal controls.

We collected 24-hour urine specimens from 23 drug-free normal controls (9 males, 14 females; mean age 28.5 ± 7.2 years) and 22 drug-free patients (7-day washout period) with panic attacks (5 males; 17 females; mean age 36.0 ± 10.5 years). All patients met DSM-III criteria for panic disorder or agoraphobia with panic attacks; controls had no present or past history of psychiatric disorder. Aliquots (50 ml) of the urine were stored frozen at −20°C until required. Tribulin was assayed by its ability to inhibit rat liver MAO activity (Glover *et al*, 1980). All urine samples were diluted with water to give a constant creatinine concentration of 30 µg/100 µl acidified to pH1 and extracted into ethylacetate. The organic phase was separated and evaporated to dryness under nitrogen. Samples were reconstituted to half of their original volume in phosphate buffer (100 mM; pH7.4). Aliquots (100 µl) of the solution were incubated with rat liver homogenate (20 µl; 2.5% w/v) and ^{14}C-tyramine (20 µl of 100 µM; specific activity 56 µCi/µmol). Control values were obtained by substituting an equivalent volume of phosphate buffer for the extracted urine specimen. Samples were incubated at 30°C for 30 min and the incubation terminated by the addition of 100 µl of HCl. The reaction products were extracted into butyl acetate and an aliquot counted. Radioactive substances were obtained from the Radiochemical Centre, Amersham, UK. Assays were performed in triplicate using analytical reagent grade chemicals from BDH, Sydney, Australia.

Triplicate determinations for individual samples varied by less than 10% of the mean value. In order to assess day-to-day variability we froze aliquots of the same sample and assessed them over 9 days. Mean MAO inhibition was 42.8 ± 4.7% of control (CV = 11%; $n = 19$). Mean MAO inhibition for the normal controls was 35.6 ± 10.2% (range = 19–49%) and for the patients was 36.2 ± 8·2% (range = 21–52%). These values are not significantly different ($P > 0{\cdot}1$; Mann-Whitney U test).

An increased tribulin output might be predicted in these patients. The failure to find the expected difference suggests that tribulin, postulated to be an endogenous anxiety-provoking agent, is not responsible for producing panic attacks. An alternative explanation might be offered by the nature of the disorder. Panic attacks are episodic, and unless urine samples were collected on the day on which a panic attack had occurred, then an elevation of tribulin output may not be detectable. Although each patient fulfilled DSM–III criteria of at least three panic attacks in the previous three weeks, we did not record if they had panic attacks on the day of urine collection. Nevertheless, panic patients do tend to have high generalised anxiety between attacks and so might be expected to produce more tribulin than controls.

To our knowledge this is the first published report of tribulin output in patients with panic. The absence of a marked difference from controls is difficult to reconcile with the putative anxiety-provoking properties of this substance(s). Studies of tribulin output in lactate-induced panic attacks might prove useful in elucidating the role of tribulin in panic disorders. The identification of the molecule and its metabolities would also clarify its role, if any, in the neurochemical mechanisms involved in anxiety states.

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