When contacted, the manufacturers (Bostik) reported that *Blu-Tack*'s constituents could give rise to glaucoma, lung damage and ultimately death if abused. In order to determine the constituents responsible for any psychotropic effect and clarify the nature of any potential toxicity, we requested an analysis of *Blu-Tack* residue from the Department of Chemistry at the University of Edinburgh. Electron ionisation mass spectrometry was unhelpful, but useful results were obtained from pyrolysis of a dried sample followed by gas chromatography separation and further mass spectrometry. A complex mixture of hydrocarbons was revealed, whose principal constituents included butene, heptadiene, propene, pentene and hexadiene. It may therefore be presumed that *Blu-Tack* smoking has attractions and risks similar to those of inhaling lighter gas, a common and well-documented phenomenon.

Blu-Tack is fairly readily available via art groups, noticeboards and wall posters even in a locked ward setting. In view of the above findings and the consequent possibility of serious risk to the physical and mental health of those attempting this activity, we wish to draw attention to it as a novel substance of abuse.

We wish to thank Professor John Monaghan (Department of Chemistry, The University of Edinburgh) for his helpful advice, and Mr W. E. Morden (I.C.I.C. & P. Ltd, Runcorn Heath, Cheshire) for performing the analysis.

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Scale for assessing hedonic tone

SIR: Snaith *et al* (1995) have developed a new pleasure scale (the Snaith Hamilton Pleasure Scale, SHAPS) corresponding to the need for a simple scale, not affected by socio-demographical factors and allowing an easy translation in other languages. They criticised the existing pleasure scales (Fawcett Clark Pleasure Capacity Scale or FCPCS; Chapman's scales) that are too long and presented cultural bias. Indeed several studies (Loas *et al*, 1992) have shown a weak discriminant validity of the FCPCS in other cultures mainly due to differences in cultural backgrounds and in socio-economic levels. Consequently we have built up (Loas *et al*, 1994) a shortened version of the FCPCS containing 12 items assessing only sensorial and physical features of pleasure. These items can be considered as being less sensitive to cultural biases.

In two studies including both healthy subjects (n=314) and patients with major depressive disorder (n=103) we demonstrated that the subscale (FCPCS-PP) has good construct and discriminant validities and a satisfactory reliability (Loas *et al*, 1994, 1995).

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Neuroleptic associated extrapyramidal symptoms

SIR: It is generally agreed that, according to the dopamine hypothesis, the antipsychotic effect of neuroleptics is mediated by blockade of mesolimbic or mesocortical D_2 dopamine receptors whereas the mechanism by which neuroleptic drugs cause extrapyramidal side-effects is not yet entirely understood. It has recently been established that Parkinson's disease, which is clinically similar to neuroleptic-induced parkinsonism, is associated with oxidative damage due to mitochondrial dysfunction (Schapira, 1994). A defect of complex I of the mitochondrial respiratory chain has been reported in platelets, muscle and brain tissue of patients with Parkinson's disease. MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) which may cause Parkinson's disease, inhibits complex I of the respiratory chain. It is a substrate for the dopamine reuptake pathway and is concentrated into mitochondria. It seemed therefore possible that neuroleptics might cause extrapyramidal side effects via a similar mechanism to that of MPTP, especially since haloperidol is structurally similar to MPTP and may be converted to a haloperidol pyridinium product analogous to MPP^+. Further evidence was derived from studies in rats which showed that neuroleptics block complex I of the respiratory chain *in vitro* (Burkhardt *et al*, 1993). We therefore

evaluated biochemically whether neuroleptics block complex I in human brain tissue. The neuroleptics studied significantly inhibited complex I in human brain tissue. The concentration of haloperidol required for half-maximal inhibition of the enzyme activity of complex I (IC_{50}) was 25 times lower than the required concentration of clozapine, and 4 times lower than that of chloropromazine. There were no significant effects of the neuroleptics on complex II and III, and on complex IV at similar drug concentrations.

The selectivity of the inhibition of respiratory chain complexes by neuroleptic drugs paralleled that induced by MPTP toxicity which leaves complexes II to IV unaffected. Furthermore, the extent of inhibition of enzyme activity parallels the incidence of extrapyramidal side-effects induced by classical *v.* the atypical neuroleptics as determined in clinical studies. The results of our study therefore support the hypothesis that extrapyramidal side-effects in humans may be the result of neurotoxicity due to oxidative damage induced by neuroleptics.

The development of tardive dyskinesias during chronic treatment with neuroleptics is often explained by the striatal dopamine receptor supersensitivity hypothesis. However, there is evidence that protracted pharmacological side-effects are not due to sustained dopamine receptor blockade. Our hypothesis is consistent with both the age-dependency of tardive dyskinesia and the higher incidence of tardive dyskinesia in patients with subtle organic changes (Pourcher *et al*, 1995). It has recently been established that with increasing age mitochondrial DNA damage increases in brain tissue (Linnane *et al*, 1989; Corral-Debrinski *et al*, 1992). Inhibition of the respiratory chain complexes induced by neuroleptics may act together with mitochondrial DNA damage due to increasing age or a preexisting reduction in oxidation due to brain lesions and further reduce oxidative phosphorylation. When a critical threshold is exceeded clinical symptoms might appear, as is established in other mitochondrial diseases.

If the antipsychotic effect and the unwanted extrapyramidal side-effects are indeed unrelated properties of neuroleptics, new principles might be applied in the development of new neuroleptics. Neuroleptics that do not damage mitochondrial respiratory chain enzyme complexes might represent a potentially important clinical advance of great benefit to the patient.

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Case reports and confidentiality

SIR: Professor Dimond's analysis (1995) appears superficial. She asserts, although without providing any supporting evidence by way of reported cases or quotations from authoritative text books, such as are normally found in legal opinions, that a breach of confidence would exist "if the patient could legitimately be identified by him/herself" as well as by others from reading a case report in a scientific journal such as the *British Journal of Psychiatry*.

A breach of confidence exists when confidential information about an identifiable individual is disclosed without proper authority. It may be legally compensatable if committed by someone who owes a duty of confidentiality to the individual concerned, and if other legal criteria are satisfied.

It is not easy to see how any breach of confidentiality could be involved in the disclosure of information about an individual to that individual himself.

The chances of anyone other than the patient himself, or a psychiatrist acquainted with him, being able to identify a patient from the sort of anonymised descriptions customarily published in psychiatric journals seem small, except perhaps in a few very unusual cases, and the chances of an action for breach of confidentiality following such an event yet more remote.

In the unlikely event of proceedings for breach of confidentiality, the plaintiff would, one imagines, need to show that he had actually been identified by at least one person other than himself, as a result of reading the report, not merely that there was a chance that that might happen.