

It is likely therefore that cognitive impairment was present in many. Hence the 'personality subtypes' identified may vanish on refeeding, or may be more pronounced, having been masked by cognitive blunting. It seems best to seek for personality subtypes in eating disorders during periods of adequate nutrition.

**Macdonald, A. J. (1995)** Eating disorders. *Journal of Family Therapy*, **17**, 356.

**Thompson-Brenner, H. & Westen, D. (2005)** Personality subtypes in eating disorders: validation of a classification in a naturalistic sample. *British Journal of Psychiatry*, **186**, 516–524.

**A. J. Macdonald** North Dorset Primary Care Trust, Forston Clinic, Dorchester DT2 9TB, UK.  
E-mail: ajmacdon@psychsft.freeserve.co.uk

**Authors' reply:** We appreciate Dr Macdonald's point that subnutrition may cause cognitive changes and other temporary conditions that may appear to affect personality. Data regarding personality in the context of adequate weight and nutrition are important for the accurate description of personality in eating disorders. It can be difficult, however, to ascertain whether shifts in personality functioning that take place through successful treatment or maturation precipitate positive nutritional changes or the reverse. Multiple studies do suggest that personality characteristics similar to those we describe in our report precede the development of eating disorders (Anderluh *et al*, 2003) and persist following remission of symptoms (Holtkamp *et al*, 2005). Although we did not report the data in detail, only 6.9% of those in our sample had a weight 15% below ideal, and the number of days of fasting was not correlated with either measure of personality pathology, suggesting this issue may not have compromised our data regarding personality to a significant extent.

**Anderluh, M. B., Tchanturia, K., Rabe-Hesketh, S., et al (2003)** Childhood obsessive–compulsive personality traits in adult women with eating disorders: defining a broader eating disorder phenotype. *American Journal of Psychiatry*, **160**, 242–247.

**Holtkamp, K., Muller, B., Heussen, N., et al (2005)** Depression, anxiety, and obsessionality in long-term recovered patients with adolescent-onset anorexia nervosa. *European Child and Adolescent Psychiatry*, **14**, 106–110.

**H. Thompson-Brenner, D. Westen** Eating Disorders Program, Center for Anxiety and Related Disorders, Psychology Department, Boston

University, 648 Beacon Street, Boston, MA 02215, USA. E-mail: ht141@hotmail.com

### Psychedelics in psychiatry

In his editorial 'Can psychedelics have a role in psychiatry once again?' (Sessa, 2005), Dr Sessa offers a detailed historical and heuristic perspective of psychedelics, with particular reference to psychotherapy. Reading the article the feeling was of sensed (by the author) repulsion of the 'neurobiological' psychiatrist in relation to 'research that explores alternative states of consciousness', 'psychedelics research' as a 'viable neurobiological substrate for the very human experience of religious encounter' and generally a possible use of psychedelics in psychiatry. Perhaps we are some of those psychiatrists who 'have been conditioned to consider such work as mysticism' but we found such a proposition challenging. We would like to discuss recent neurobiological findings related to one of the psychedelics mentioned by Dr Sessa, which perhaps would offer an explanation as to why these substances have limited scope in psychiatry today.

3,4-Methylenedioxymethamphetamine (MDMA), also known as ecstasy, is largely consumed by young adults as a recreational drug. Common doses of this popular compound (60–120 mg, equivalent to 1–2 tablets) produce unexpectedly high blood levels, with MDMA present at high concentration at the receptor level. The drug induces dose-dependent neurotoxicity in animal models and humans; this mainly involves the central serotonergic system (Ricaurte *et al*, 2002). Serotonin is important for brain development and maintenance of neural and glial function in the mature brain (Azmitia, 2001). Another interesting mechanism involves the 'pruning' of serotonergic neurons (Ricaurte *et al*, 2000). The drug appears to reduce the number of serotonin axons and axon terminals but nerve cells will often replace terminals upstream for the damaged ones. The resulting effect is of substantial impaired connectivity. Younger brains are particularly susceptible because of increased neuroplasticity, resulting in a substantial reorganisation of brain connectivity.

Functional magnetic resonance imaging studies suggest a decreased activation in inferior temporal regions, the hippocampus, angular gyrus and striate cortex

associated with working memory performance (Daumann *et al*, 2003, 2005), with the hippocampus and globus pallidus being possibly more sensitive (Reneman *et al*, 2001; Jacobsen *et al*, 2004; Daumann *et al*, 2005). More recent voxel-based morphometry studies support the hypothesis that the use of MDMA leads to reduction in cortical grey matter in multiple brain regions, including the neocortex, brain-stem, cerebellum and anterior cingulate gyrus, reflecting compromised serotonergic activity (Cowan *et al*, 2003). Although we have mentioned only a few studies, there is substantial evidence to suggest considerable neurotoxicity of compounds such as those mentioned by Dr Sessa. There are concerns about possible long-term adverse effects of psychedelics in both infrequent and regular users, which explain why psychiatrists are reluctant to consider such substances in their pharmacological armamentarium.

**Azmitia, E. C. (2001)** Modern views on an ancient chemical: serotonin effects on cell proliferation, maturation, and apoptosis. *Brain Research Bulletin*, **56**, 413–424.

**Cowan, R. L., Lyoo, I. K., Sung, S. M., et al (2003)** Reduced cortical gray matter density in human MDMA (Ecstasy) users: a voxel-based morphometry study. *Drug and Alcohol Dependence*, **72**, 225–235.

**Daumann, J., Schnitker, R., Weidemann, J., et al (2003)** Memory correlates of working memory in pure and polyvalent ecstasy (MDMA) users. *Neuroreport*, **14**, 1983–1987.

**Daumann, J., Fischermann, T., Heekeren, K., et al (2005)** Memory-related hippocampal dysfunction in poly-drug ecstasy (3,4-methylenedioxymethamphetamine) users. *Psychopharmacology*, **180**, 607–611.

**Jacobsen, J. K., Mencl, W. E., Pugh, K. R., et al (2004)** Preliminary evidence of hippocampal dysfunction in adolescent MDMA ('ecstasy') users: possible relationship to neurotoxic effects. *Psychopharmacology*, **173**, 383–390.

**Reneman, L., Majoie, C. B., Habraken, J. B., et al (2001)** Effects of ecstasy (MDMA) on the brain in abstinent users: initial observations with diffusion and perfusion MR imaging. *Radiology*, **220**, 611–617.

**Ricaurte, G. A., Yuan, J., McCann, U. D. (2000)** ( $\pm$ )3,4-Methylenedioxymethamphetamine ('Ecstasy')-induced serotonin neurotoxicity: studies in animals. *Neuropsychobiology*, **42**, 5–10.

**Ricaurte, G. A., Yuan, J., Hatzidimitriou, G., et al (2002)** Severe dopaminergic neurotoxicity in primates after a common recreational dose regimen of MDMA ('ecstasy'). *Science*, **297**, 2260–2263.

**Sessa, B. (2005)** Can psychedelics have a role in psychiatry once again? *British Journal of Psychiatry*, **186**, 457–458.

**D. Arnone, F. Schifano** Department of Mental Health – Addictive Behaviour, St George's University of London, Cranmer Terrace, London SW17 0RE, UK. E-mail: darnone@sgul.ac.uk