to 'limitations' in both MRI and metaanalysis. The authors are right to highlight the problem of anatomical definition of the amygdala *in vivo* and how other imaging parameters may obscure (or reveal) laterality effects and differences between subject groups. However, they are wrong to blame meta-analysis. Systematic review and meta-analysis of MRI data is a powerful means of quantifying the precise effects that are the subject of speculation by Chance and colleagues.

We have recently carried out just such a review of the normal human amygdala (Brierley et al, 2002). Some 39 studies and 51 data-sets met our inclusion criteria, allowing comparison of 1491 amygdala pairs. The weighted mean volumes (95% CI) for the left and right amygdala were (35.1) and 1691.7 mm³ $1726.7 \, \text{mm}^3$ (37.2), respectively. The range was from 1050 to 3880 mm³. This variance is clearly worrying. We were able to examine systematically some of the causes of this and found that most imaging parameters, such as slice thickness and plane of orientation, were not particularly influential. Study size had a weak but significant effect, with larger studies tending to give smaller volumes. Anatomical definition was the most important variable. Studies which employed the Watson criteria (Watson et al, 1992) gave significantly larger volumes than the remainder. Gender differences persisted (male greater than female) even in studies which attempted to control for intracranial volume. Laterality effects were not significant.

The ease of obtaining high-resolution anatomical brain images afforded by modern MRI on large samples of individuals across the life span means that MRI should be taken as the gold standard on regional volumetrics rather than post-mortem samples with all their attendant deficiencies. However, in order to exploit the advantages of MRI, researchers must pay particular attention to reliability of anatomical definitions. We have proposed that Watson's criteria be adopted generally and have recommended some minor improvements (Brierley et al., 2002).

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Authors' reply: We agree with David et al that the key problem, which we have highlighted, in in vivo MRI studies of the amygdala, is anatomical definition. The ability to define anatomical boundaries at the cellular level means that post-mortem samples set the gold standard for anatomical delineation. Indeed, the generally smaller volumes of the amygdala (uncorrected for tissue shrinkage in Chance et al, 2002) reported in post-mortem studies are indicative of more conservative estimates when the precise boundaries can be seen. This is consistent with Brierley et al's (2002) conclusion in their meta-analysis that anatomical definition is the most prominent contributor to variance in MRI volume estimates of the amygdala.

Our criticism is not of meta-analysis per se, but of the inclusion of some studies, which owing to low scan resolution use only very approximate anatomical definitions. Particularly problematic in schizophrenia is the use of landmarks, which may be systematically shifted with respect to the boundary of the amygdala, owing to other changes in the temporal lobe. While MRI studies have the obvious advantages of an in vivo assessment and larger sample size, post-mortem studies are also important as a check on the possibility of systematic bias which may enter the MRI literature (Walker et al, 2002).

We agree with the importance of consensus criteria for anatomical definitions which take full advantage of the improvements in up-to-date MRI visualisation. Our paper concludes with some references to studies attempting to tackle this issue for the amygdala, to which the paper of Brierley *et al* (2002) should be added.

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Phenomenology of acute confusional states

I read with great interest the paper by Dr Fleminger (2002) on delirium, and the relevant controversy raised by Dr Philpott regarding to whom should be attributed the first description of hypoactive delirious states (Philpott, 2002). May I suggest that this initial description was made around one century earlier than mentioned by both authors. In fact, as early as 1892 the French alienist Philippe Chaslin borrowed the term of 'confusion mentale primitive' from a previous description proposed by Delasiauve during the 1850s. He was probably one of the first authors who gathered under a unified entity what was previously described under separate clinical features as psychosis post-influenza, post-acute diseases, postfever and epilepsy (Chaslin, 1892). He also clearly noticed its similarity with what Lasegue had described earlier as delirium tremens, in which perceptual disturbances were considered as a dream-like experience (Lasegue, 1881). In his later monograph, Chaslin describes the acute confusional state as 'an acute brain disorder, consecutive to an organic significant disease, with cognitive impairment associated with delusions, hallucinations, psychomotor agitation, or reciprocally, with psychomotor retardation and inertia' (Chaslin, 1895). Despite this very early description of what has since been called hyperactive and hypoactive subtypes of delirium, there have been very few attempts to test the validity and the relevance of these subtypes. To our knowledge, at this time only one empirical exploration of what are the constitutive symptoms of each dimension has been proposed (Camus et al, 2000). We would like to add, concerning what Fleminger cites as possible psychological consequences of confusional experience, that another French alienist described 'permanent ideations' and 'chronic delusional states' following the post-dream-like confusional experience (Regis, 1911). We agree with Fleminger's assumption that hyperactive subtypes are among the most stressful confusional experiences because of the possible persistence of memories of perceptual disturbances beyond the full recovery of consciousness and arousal, and beyond the normalisation of the sleep-wake cycle. But it remains unclear what factors are associated with such persistent difficulties in overcoming the dream-like experience. We hypothesise that they could be related to the implication of some specific neurobiological pathways, but their potential relationship with some premorbid personality traits should also be explored. Finally, as long as the pathophysiology of delirium is poorly understood, research into biological markers such as cerebrospinal fluid levels of neuropeptides (Broadhurst & Wilson, 2001) should be correlated to all different aspects of delirium phenomenology.

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Psychiatry in China

In response to Lyons (2001) and Kumar (2000), I have been working on the research of *Qigong*-related mental disorder and culture-bound syndrome in China for over a decade and I feel it is unfair for psychiatry in China to be represented by their remarks. I would argue that it is the misuse of *Qigong*, rather than misuse of psychiatry, that is at issue in China, according to my experience of research of *Falun Gong*-related mental disorder and culture-bound syndrome (Shan *et al.*, 1987, 2000; Shan,

1999). Some of the reports about the abuse of psychiatry in China are based on political issues and lack any awareness of academic research and study in China. In fact, *Qigong* was misused in China, and the patients and practitioners who suffered with *Falun-Gong*-related mental disorders need to be treated in psychiatry. I must call for more experts in psychiatry and in the World Psychiatric Association to pay attention to the research of *Qigong*- and *Falun-Gong*-related mental disorders.

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One hundred years ago

Amaurotic family idiocy

Amaurotic family idiocy is a rare condition which has been observed in Jewish children. It was first described in 1871 by Mr. Waren Tay; since then about 68 cases have been recorded. In the New York Medical Journal of July 12th Dr A. Hymanson has published the following case. A male infant whose parents were Russian Jews appeared normal until the eighth month, when he ceased to take any interest in his surroundings. He would not raise his head and made purposeless movements of the limbs. The head was large, measuring 19 inches in circumference. The fontanelles were prominent and widely open, but at the age of 10 months they were gradually closing. At the age of 15 months he could not hold his head up; it was usually thrown backwards. He was very anaemic, his muscles daily became weaker and more flabby,

and spontaneous movements gradually ceased. He had a vacant look and seemed to see light but did not recognise his parents. He seemed to be deaf but became frightened when anyone knocked at the door. During sleep the eyes and mouth were wide open. The pupils were slightly contracted and did not react to light. The fundi showed changes exactly similar to those described by Mr. Waren Tay. Corresponding to the macula lutea of each eye was a large bluish-white spot about twice the size of the optic disc. At its centre was a brownish-red circular dot. The optic discs were in a state of grey atrophy and the calibre of the vessels was markedly reduced. The child died at the age of 19 months. Two weeks before death there was great anorexia. He was much emaciated, could hardly move his limbs, and had gluteal bed-sores. The temperature was subnormal, 97.5° to 98° F. A necropsy was refused. Of the 68 recorded cases 40 are known to have been fatal: the result in the others is unknown. The family predisposition is shown by the fact that 28 cases occurred in 18 families. 30 cases were observed in America, 11 in England, 14 in Germany, and the remainder in other countries. The necropsies have not shown any abnormality in the form or structure of the cerebral convolutions. Thus the etiology and pathology are unknown. The chief clinical features are idiocy, weakness of all the muscles terminating in paralysis, gradual loss of sight, characteristic changes in the macula lutea, marasmus, and death at the end of the second year.

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