Table 1 D_2 dopamine receptor binding indices in striatum, thalamus and temporal cortex, and the ratios of temporal/striatal (temporo-striatal) and thalamic/striatal (thalamo-striatal) binding indices in patients taking traditional and atypical antipsychotics (data from Xiberas *et al*, 2001)

Drug	Binding index (%)			Temporo-striatal index	Thalamo-striatal index
	Striatum	Thalamus	Temporal cortex		
Haloperidol 3 mg	66.6	91.2	88.3	1.33	1.37
Risperidone 6 mg	67	92.2	92.2	1.38	1.38
Amisulpride 1000 mg	61.5	69.9	87.8	1.43	1.14
Olanzapine 20 mg	69.6	91.9	91.8	1.32	1.32
Clozapine 200 mg	45.9	79	90.1	1.96	1.72

blockade in temporal cortex caused by atypical antipsychotics (Pilowsky *et al*, 1997; Bigliani *et al*, 2000).

Looking at the data from Xiberas *et al* (2001), we came to different conclusions. Using equipotent doses of antipsychotics (doses which lead to the same occupation of D_2 receptors in the striatum), no differences in thalamo-striatal and temporo-striatal indices between typical and atypical antipsychotics could be shown (Table 1). We suggest that atypical antipsychotics do not exert special temporal lobe or limbic selectivity. The selectivity depends more on the dose than on the type of antipsychotic (typical *v*. atypical). This is in agreement with Nyberg & Farde (2000) and Geddes *et al* (2000), who argue that non-equipotent doses can partly explain differences between classical and novel antipsychotics.

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Authors' reply: We thank Dr Kopeček *et al* for their interest in our paper (Xiberas *et al*, 2001*b*). They conclude that atypical antipsychotics do not exert special temporal or limbic selectivity, which depends instead on drug dosages. First, we believe that generalisations drawn from data obtained from five patients, each one treated with a different antipsychotic drug, are not sound, because of intersubject variability. For instance, should Dr Kopeček *et al* have considered plasma drug concentrations and patient H2 of our article, their conclusion would have been modified. In our article, we drew conclusions from the statistical comparisons of [^{76}Br]-FLB457 measures obtained with positron emission tomography (PET) in subgroups of patients, receiving the usual dosage recommended by the pharmaceutical firms for each antipsychotic drug, for treating psychotic episodes.

Second, we have already reported the importance of dosage when interpreting neuroimaging measures of regional D_2 dopamine receptor blockade by antipsychotic drugs (Xiberas *et al*, 2001*a*). Inspection of the table that Kopeček *et al* draw from our article suggests that for a striatal D_2 receptor binding index approaching 65–70%, the atypical antipsychotics induce extrastriatal/striatal indices comparable with that induced by the lowest oral dosage of haloperidol reported. This is consistent with our previous publication (Xiberas *et al*, 2001*a*) where we specifically highlighted the dose-dependence of extrastriatal/striatal D_2 blockade, from a study in a larger sample of patients treated with an atypical antipsychotic. We demonstrated that plasma concentrations were more accurately related than daily oral doses to the different regional binding profiles determined with PET. Clearly, two binding profiles could be distinguished depending on the plasma concentration of the drug: low striatal binding associated with marked extrastriatal binding for low plasma concentrations, or marked binding in both striatal and extrastriatal regions for higher plasma concentrations. This may be applicable to both atypical and typical compounds, if very low doses of typical neuroleptics (i.e. below the recommended therapeutic dose range) are considered, but this is a speculation. Therefore, having previously highlighted the effect of dosage (Xiberas *et al*, 2001*a*), we chose to highlight in our second article (Xiberas *et al*, 2001*b*) that, at plasma concentrations obtained in actual clinical practice, and compared with haloperidol, various atypical antipsychotic drugs have a regional binding profile that is higher in mesocorticolimbic regions than in striatum.

Declaration of interest

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Xiberas, X., Martinot, J. L., Mallet L., *et al* (2001*a*) *In vivo* extrastriatal and striatal D_2 dopamine receptor blockade by amisulpride in schizophrenia. *Journal of Clinical Psychopharmacology*, **21**, 207–214.

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Measuring amygdala volume

Chance *et al* (2002) described volumetric measurement of the amygdala and found few differences between normal and schizophrenia post-mortem samples. This fails to confirm published magnetic resonance imaging (MRI) data on hundreds of individuals which have been systematically reviewed and analysed (Wright *et al*, 2000). Chance *et al* (2002) report mean absolute volumes (643 mm^3 for nine men and 612 mm^3 for nine women) that are much smaller than those reported in MRI studies. They go on to speculate on the reasons for this discrepancy and point

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