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 D2 receptors in the striatum), no differences in thalamo-striatal and temporostriatal 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 vs. 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 Kopecek et al for their interest in our paper (Xiberas et al., 2001b). 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 Kopecek 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 [123I]epidepride single photon 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 D2 dopamine receptor blockade by antipsychotic drugs (Xiberas et al., 2001a). Inspection of the table that Kopecek et al draw from our article suggests that for a striatal D2 receptor binding index approaching 65–70%, the atypical antipsychotics induce extra-striatal/striatal indices comparable with that induced by the lowest oral dosage of haloperidol reported. This is consistent with our previous publication (Xiberas et al., 2001a) where we specifically highlighted the dose-dependence of extra-striatal/striatal D2 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., 2001a), we chose to highlight in our second article (Xiberas et al., 2001b) 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**

Our team is funded by a donation from the INSERM. Also X.X. was partly funded by grants from the Fondation pour la Recherche Médicale and the Commissariat à l’Energie Atomique.


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**CORRESPONDENCE**

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 mm3 for nine men and 612 mm3 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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**Table 1** D2 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)

<table>
<thead>
<tr>
<th>Drug</th>
<th>Binding index (%)</th>
<th>Temporo-striatal index</th>
<th>Thalamo-striatal index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Striatum</td>
<td>Thalamus</td>
<td>Temporal cortex</td>
</tr>
<tr>
<td>Haloperidol 3 mg</td>
<td>66.6</td>
<td>91.2</td>
<td>88.3</td>
</tr>
<tr>
<td>Risperidone 6 mg</td>
<td>67</td>
<td>92.2</td>
<td>92.2</td>
</tr>
<tr>
<td>Amisulpride 1000 mg</td>
<td>61.5</td>
<td>69.9</td>
<td>87.8</td>
</tr>
<tr>
<td>Olanzapine 20 mg</td>
<td>69.6</td>
<td>91.9</td>
<td>91.8</td>
</tr>
<tr>
<td>Clozapine 200 mg</td>
<td>45.9</td>
<td>79</td>
<td>90.1</td>
</tr>
</tbody>
</table>

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