manage to cope and keep working after many stressful life events, and yet have work disability and develop post-traumatic stress disorder after minor motor vehicle collisions. Perhaps it is as Sir John Collie remarked long ago:

> 'In short, the essential quality of a thing is its worth to the individual, and its value to him is its power to serve his private ends. On one occasion, when examining a working-man for an injury to his thumb, he observed me examining the terminal phalanx of one of his fingers, which had been partially removed, obviously as the result of a former accident. "That," said he, "is of no importance; it was done at home"!' (Collie, 1917).

Collie, J. (1917) *Malingering and Feigned Sickness* (2nd edn), p. 15. London: Edward Arnold.

Karlsborg, M., Smed, A., Jespersen, H., *et al* (1997) A prospective study of 39 patients with whiplash injury. *Acta Neurologica Scandinavica*, **95**, 65–72.

Mayou, R. & Bryant, B. (2002) Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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Regarding Mayou & Bryant's study (2002), it is interesting that the predictors of pain at 1 year are 'feeling not to blame for the accident', claiming compensation and 'anger cognitions'. With multivariate analysis, only claiming compensation at 3 months is a predictor of pain at 1 year. This means that feeling not to blame for the accident, initial anger or anger cognitions are predictors of pain only in claimants, otherwise not. Thus, of two patients, both not-at-fault, and both equally angry, it is the one who chooses litigation that will have the worse outcome. Why?

Does litigation/claims create a psychosomatic phenomenon that allows anger and victimisation to express itself as pain? Or are litigants more likely to be compelled to focus on all sources of aches and pains in their life (even pre-accident sources) by keeping pain diaries more often and by being instructed to see more physicians and therapists, to withdraw from more activities that hurt, to take more medications, to develop poor physical fitness, postural problems, medication adverse effects and anxiety?

It is further interesting that 14% of accident victims with no injury had bodily pain at 3 months! How does this happen? Is it a manifestation of psychological distress, or perhaps does pain occur as part of life, even if not injured (or, for that matter, even if not involved in an accident)? The percentage of accident victims with pain at 1 year in the 'no injury' group is half that of whiplash injury victims with pain at 1 year (27%). As one does not expect whiplash injury to create an immunity from whatever is affecting the 'no injury group', half of the whiplash injury group was going to have pain at 1 year, even if they had had no injury, or had fully recovered from their injury, because the 'no injury' group gets pain anyway. Not all of the pain at 1 year in whiplash victims can thus be due to physical effects of the initial injury, since then there would be at least some additional burden of pain from whatever factors also cause pain in the 'no injury' group as well. Statistically, half of the chronic pain that exists in whiplash patients is independent of having had an initial physical injury.

The findings of this study also suggest that when a physician encounters a patient who is not to blame for an accident and who is feeling angry, the physician should very clearly advise that entering a claim will adversely affect the patient's health and is more likely to lead to chronic pain.

Mayou, R. & Bryant, B. (2002) Psychiatry of whiplash neck injury. *British Journal of Psychiatry*, **180**, 441–448.

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Authors' reply: Drs Kwan and Friel make familiar general points about the interpretation of prospective research. However, they underestimate the practical, methodological and ethical difficulties of obtaining and using medical records and of qualifying information about life events. It is also worth noting that in medico-legal practice it is very common for medical experts and lawyers to disagree about the significance of medical histories and of life events following the identified trauma.

Dr Ferrari's first paragraph over-interprets multivariate analysis dependent on statistical significance in concluding that initial anger or anger cognitions are early predictors of pain in claimants. Although there are some differences between claimants and non-claimants, our overall experience in this study, and in a previous paper which followed up claimants for 6 years, is that the two groups are very similar (Bryant *et al*, 1997). The research findings, together with clinical experience, indicate that litigation is one of a number of reminders of the accident which do result in subjects focusing on their aches and pains. Further accidents, continuing medical complications and persistent financial difficulties are probably other important factors acting in a similar manner.

Fourteen per cent of accident victims with no recorded injury in the emergency department had pain at 3 months which was attributed to the accident. Perhaps the most likely explanation is that these people suffered minor musculo-skeletal injuries but that the symptoms did not become significant for hours or days after the accident. This is well described in relation to whiplash neck injury. It is therefore incorrect for Dr Ferrari to use our evidence to draw conclusions about the extent to which pain reported by whiplash patients may be independent of physical injury.

I also strongly disagree with Dr Ferrari's final conclusion that patients who are not to blame but angry should be advised not to enter a claim. The financial and other losses may be considerable and compensation desirable and even necessary. The more appropriate conclusion is that medical and legal procedures should take account of the patient's reactions and beliefs, avoid increasing distress and attempt to provide a sympathetic and rapid resolution of both the medical and the legal issues.

Bryant, B., Mayou, R. & Lloyd-Bostock, S. (1997) Compensation claims following road accidents: a six-year follow-up study. *Medicine, Science and the Law*, **37**, 326–336.

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Regional selectivity of novel antipsychotics

Xiberas *et al* (2001) measured D_2 receptor occupancy in striatum, thalamus and temporal cortex in patients treated with haloperidol, risperidone, amisulpride, clozapine and olanzapine. On the basis of their findings, they conclude that in the striatum and in the thalamus atypical antipsychotics induce a significantly lower D_2 binding index than haloperidol does. Their results are consistent with previous studies showing only small differences between striatal and temporal cortex blockade by traditional compounds and relatively selective D_2

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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.

Bigliani, V., Mulligan, R. S., Acton, P. D., *et al* (2000) Striatal and temporal cortical D2/D3 receptor occupancy by olanzapine and sertindole *in vivo*: a [123I]epidepride single photon emission tomography (SPET) study. *Psychopharmacology*, **150**, 132–140.

Geddes, J., Freemantle, N., Harrison, P., *et al* (2000) Atypical antipsychotics in the treatment of schizophrenia: systematic overview and meta-regression analysis. *BMJ*, **321**, 1371–1376.

Nyberg, S. & Farde, L. (2000) Non-equipotent doses partly explain differences among antipsychotics – implications of PET studies. *Psychopharmacology*, **148**, 22–23.

Pilowsky, L. S., Mulligan, R. S., Acton, P. D., *et al* (1997) Limbic selectivity of clozapine. *Lancet*, **350**, 490–491.

Xiberas, X., Martinot, J. L., Mallet, L., *et al* (2001) Extrastriatal and striatal D_2 dopamine receptor blockade with haloperidol or new antipsychotic drugs in patients with schizophrenia. *British Journal of Psychiatry*, **179**, 503–508.

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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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