Autism is caused by impairment in the brain, not the gastrointestinal tract, and probably not that much by genes either; several reports of identical twins discordant for autism have been published.[5] The impairment in the brain that prevents normal language development should be the focus of research, and how such impairment can come about. Brainstem damage caused by oxygen deprivation at birth deserves attention as a reasonable starting point, especially if funds are short, before more esoteric causes are considered.

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A prisoner with acute renal failure

Sir—Doris Chan and colleagues' Case report (Jan 10, p 126)[1] is a cautionary tale of the folly of treating alcohol withdrawal seizures with standard anticonvulsant medication.

The treatment of multiple alcohol withdrawal seizures with an oral or a parenteral short-acting benzodiazepine is an established evidence-based protocol.[2] The only long-term management consideration is a rule-out work-up for other causes of seizures—ie, central nervous system tumours or other space-taking lesions. Once satisfied that the patient has had a withdrawal seizure, the interdiction of alcohol consumption is the treatment. In fact, there might be evidence that standard antiseizure medication (phenytoin, carbamazepine, etc) could actually kindle alcohol withdrawal seizures.[2]

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Unprotected sex and alloimmune activation

Sir—Barry Peters and co-workers (Feb 14, p 518)[1] report that unprotected sex results in alloimmune activation and in reduced susceptibility to HIV-1. This conclusion was drawn from the results of mixed leucocyte reactions (MLRs) and from in-vitro HIV-1 infectivity studies of CD4+ T cells in heterosexual couples having unprotected sex and in women and men having protected sex.

First of all, we believe that some of the data are not correctly presented. Table 2 is meant to show MLR stimulation indices from couples having unprotected sex in comparison with such indices from women and men having protected sex after stimulation with control cells from third-party men and women. However, the data for women and men having unprotected sex are not those after stimulation with control cells, but after stimulation with their partner's cells. This fact can be deduced from table 1 in which the same data are presented. When comparing the correct control-cell-stimulated MLR data from women and men having unprotected sex (table 1) with women and men having protected sex (table 2), no differences are apparent.

We think that the important question of whether unprotected sex results in a significantly higher alloimmune response than protected sex for a time remains unanswered. To test this theory, the authors should have compared MLR data after stimulation with the partner's cells rather than using control cells for both the study groups. Unfortunately, this could not be done because previous sex partners of women and men who were practising protected sex at the time of the study were not available. Subdividing this study group into participants who had been practising protected sex for 6–12 months and more than 12 months to control for the duration of the putative alloimmune response elicited by previous unprotected sex does not solve this problem. The observation that women and men having unprotected sex generate a significantly higher alloimmune reaction against their partner's cells than against control cells (table 1 and figure 1) does not provide evidence on its own. A similar difference among women and men who are having protected sex for 6–12 months or more than 12 months might have persisted over time; however, this analysis was not done.

The significantly lower in-vitro susceptibility of CD4+ T cells to HIV-1 in women and men having unprotected sex than those having protected sex is intriguing. For the reasons described above, we believe that whether an increased alloimmune reaction is responsible for the observed in-vitro HIV-1 resistance remains unproven. Furthermore, in-vitro HIV-1 infection was done with phytohaemagglutinin-activated CD4+ T cells, which might not represent the susceptibility of allostimulated target cells. Thus, the possibility of tolerance towards alloantigens as a consequence of unprotected sex might not be excluded by the present data, in line with maternal tolerance towards the fetus as the result of previous exposure to paternal alloantigens via unprotected sex,[2] previous pregnancies,[3] or blood transfusions.[4]

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Authors' reply

Sir—Our major aim was to determine whether HLA antigens in seminal fluid elicit alloimmunity or indeed tolerance in women practising unprotected sex with regular partners. We found that 23 of the 29 women and men showed significantly higher MLRs to the partner's than to unrelated control cells, consistent with alloimmunity, and six showed the converse results, raising the possibility of tolerance. These were surprising findings that might be important in our understanding of genital immunity.

Wim Jennes and Luc Kestens' point about the confusing labelling of part of table 2 is correct, since comparison was clearly made between MLRs of cells from women having unprotected sex stimulated with their partners' cells, and cells from those having protected sex stimulated with unrelated control cells. We stated this in both the Results and Discussion, and a comparison of MLRs between women practising unprotected