symptomatology, with extremely traumatic situations such as physical and sexual abuse being related to very high PTSD symptomatology scores. However, the number of participants with exposure to such traumatic events was very small (n=9 for physical abuse, n=4 for sexual abuse and n=13 for physical and sexual abuse as a child).

Notwithstanding, the basic message of the paper is important: the line between life events and traumatic events is at best thin, and sometimes nonexistent. The best support for this can be found in the case of the Dutch farmers (Olff *et al*, 2005) whose cattle were exposed to foot and mouth disease leading to the killing of the herds. This was not a life-threatening event for the farmers, but was a major life event that can easily be considered traumatic.

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Post-partum depression

We would like to raise some concerns about the paper by Evans *et al* (2005), which oversimplifies the aetiology of post-partum depression. Depression in pregnancy and post-partum has been globally linked to psychosocial issues (marital problems, social support, childhood adversity) and pregnancy-related factors, all of which interact with personality (Patel *et al*, 2002; Dennis & Boyce, 2004). For the findings of the paper to be clinically relevant, it would have been useful to study the relative roles of at least some of these mediating variables, rather than focusing on personality alone.

We also feel that using six items from an interpersonal sensitivity scale for assessing the main explanatory variable is not fully justified. The items chosen measure only some aspects of the self; more-robust measures such as the Dysfunctional Attitude Scale (Weissman, 1979), or the Crandell Cognitions Inventory (Crandell & Chambless, 1986) could have been used to assess self-schemas.

We would also like Evans *et al* to speculate on why some women developed depression earlier and some later (after 3 years) despite having high negative self-schemas at baseline. Is it possible that self-schema also change with experiences such as motherhood, or that support might have mediated the later onset of depression? Also, did women in the higher tertiles for negative self-schema score develop depression earlier?

In the absence of information about important psychosocial variables and factors related to the development of schemas, it is difficult to presume that negative self-schemas are alone sufficient to predict the onset of depression. The inclusion of women who had negative self-schemas but did not develop depression, and repeat assessment of those with negative self-schemas would have also better delineated state versus trait concerns. Finally, it would have been useful to have a control group of non-pregnant women to determine whether personality as a vulnerability factor is unique to pregnancy and the post-partum period.

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Authors' reply: Drs Chandra and Sudhir appear to have misunderstood the aim of our paper. The paper is not primarily about post-partum depression. We did not aim to study the overall aetiology of post-partum depression nor did we aim to accurately predict post-partum depression from the negative self-schema measure. We did aim to test an important aspect of the cognitive theory of depression, namely whether a measure of negative self-schema is associated with the onset of depression. We found there was an association and that it was equally strong whether the onset was during pregnancy, in the post-partum period or 3 years later. In the main analysis we adjusted for the psychological and socio-economic variables outlined in Table 3.

We agree, as stated in our discussion, that a more detailed questionnaire such as the Dysfunctional Attitude Scale would have provided a more comprehensive measure of self-schema. Furthermore, repeated measures would have allowed comparison with other studies and a test of the stability of these 'schemas'. It is possible that schemas change with an experience such as motherhood, although theoretically they should be relatively stable. As these were secondary data analyses of an existing data-set, we were limited to the data available to us and these did not include any more-detailed or repeated measures of schemas.

There are clearly multiple factors that influence the onset of depression. The correspondents ask why some women have earlier onset than others. This may well be related to changing support or adverse events, but it was not the aim of our paper to address this question. Rather than speculate, the ALSPAC data-set provides an opportunity to answer this question and many others by undertaking further detailed analyses of those data.

The analyses we presented in Table 4 indicate that the strength of the association between negative self-schema and onset of depression does not diminish with time, so it is unlikely that those in the highest tertiles for depression have onset which is sooner.

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This work needs to be replicated in other large longitudinal studies of both non-pregnant women and men.

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Hypertension and depression in late life

The 'vascular depression' hypothesis has attracted considerable interest, but its basis is by no means clear. Kaimal & Nair (2005) in their recent correspondence mention vascular comorbidity in late-onset depression, citing a high prevalence rate of hypertension in particular. However, despite reasonably consistent findings of neuroradiological abnormalities associated with late-life depression, there is actually little evidence that hypertension or other 'traditional' vascular risk factors are responsible. What evidence there is for higher comorbidity comes from case–control studies comparing people with late-life depression who are known to clinical services with community controls; these studies carry a high risk of selection bias. Studies carried out in community populations have not generally found any associations between hypertension and depression (Kim *et al*, 2004), even in high-risk samples (Stewart *et al*, 2001).

The direction of causation between vascular disorders and depression is also unclear (Baldwin, 2005). Evidence for neuroradiological abnormalities associated with depression is derived almost entirely from cross-sectional studies, and there is currently much stronger prospective evidence that depression is a risk factor for vascular disorders than vice versa. The same appears to be the case for sub-syndromal depressive symptoms that may not be recalled or reported in later life and might well have been present in people whose depression is classified as 'late-onset'. Neuropathological studies of late-life depression do not find the micro-vascular abnormalities that would be expected if hypertension were responsible, but instead find increased large vessel disease (Thomas *et al*, 2001), which could equally be a consequence as a cause of depression.

The uncertainty surrounding specific vascular processes in late-life depression is not just of academic interest, since there are important public health implications. Depression is strongly predicted by poor physical health and associated disability and there is little evidence at present to justify distinguishing specific disorders as responsible. The relationship between vascular processes and affective disorder is interesting, but there is a danger that more obvious risk factors for late-life depression (disability, poverty, loneliness) are ignored because they do not fall within the exciting world of organic psychiatry.

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Detection of depression in elderly care home residents

Dr Eisses *et al* (2005) showed how the training of care home staff can help in detecting depression among elderly care home residents. In view of the high prevalence of depression in this group, these results are to be welcomed. However, I would like to raise a few points about the relevance of this report to care homes in England.

First, 8 out of 23 homes (35%) declined to participate, citing lack of interest or feeling that it was too much work. This matches my own experience as a consultant old age psychiatrist dedicated to providing support to care homes with nursing. I would welcome any suggestions on how to engage homes in training to improve the detection of depression in this vulnerable population.

Second, I was impressed by the stability of the workforce, who had spent on average 9.5 years in the participating homes (the shortest period being 10 months). This is considerably longer than is found within some care homes in England, particularly in the large cities. It may be that training would be less effective when staff tend to have a higher rate of turnover.

Finally, I note that the study excluded residents with dementia who scored less than 15 out of 30 on the Mini-Mental State Examination. This decision is understandable as the scale used, the Geriatric Depression Scale (GDS), is difficult to administer to this group. However, I wonder about the effect on the staff. Training staff to exclude these residents from an assessment of depression could send a false signal that these individuals are somehow immune to depression – which most certainly is not the case. Also this approach probably could not apply in care homes with nursing in England. Whereas in the homes studied the prevalence of dementia was only some 9% of the population, dementia in care homes with nursing is high, with 'non-EMI' (elderly mental illness) care homes with nursing having a prevalence of dementia as high as 75% (Macdonald & Carpenter, 2003). We use the Cornell Scale for Depression in Dementia (Alexopoulos *et al*, 1988) for those people with dementia who can not respond to the questions of the GDS.

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