We agree with Macdonald that the CRP serum levels may help to focus clinical attention on the diagnosis and the management of delirium. However, although CRP may be viewed as a measure of inflammatory activity, it may not be forgotten that it actually captures only one specific aspect of inflammation, which is not necessarily the most relevant for delirium [4].

**Data are expressed as means ±SD unless otherwise specified;**

**a** for patients with prevalent delirium, C-reactive protein serum levels at delirium onset were not available.  

**Delirium and C-reactive protein**

SIR—The paper by Macdonald et al. [1] suggests that C-reactive protein (C-RP) independently predicts incident delirium and recovery. This finding is based on a sample of 86 acutely ill patients aged 70 years or more, of whom 26 had prevalent delirium but only six developed incident delirium. The authors comment on the limitations of results based on small samples and the need to replicate their findings. We tried to do so using data from a larger prospective observational study of 283 unsolicited medical emergency patients aged 75 years or more, admitted within 24 h of admission in a district general hospital. Of these, 76 patients had prevalent delirium [identified by Confusion Assessment Method (CAM) and fulfilling DSM-IV criteria] and 29 developed incident delirium. C-RP levels on admission were available for 81% of patients.

As previously reported [2], we found significantly higher levels of C-RP on admission in those with prevalent delirium (88.8) (mean ± SD) or incident delirium (28.6) compared to those who never developed delirium (31.9 mg/l ± 101.5). However, there was no difference between the prevalent and incident delirium groups (P = 0.83). In eight patients with incident delirium, C-RP levels measured at the time of onset of delirium did not differ significantly from those on admission (P = 0.091). We found no association between admission C-RP level and delirium duration (r = 0.041, P = 0.674) or delirium severity measured using the Delirium Assessment Scale [3] (r = 0.111, P = 0.286). Using a similar definition of recovery from delirium as Macdonal et al. (a CAM-positive assessment followed by CAM negatives until discharge) all but a single case of delirium recovered.

**Letters to the Editor**

**Table 1.** Characteristics of 110 patients with delirium admitted to a rehabilitation and aged care unit, according to the types and the causes of delirium

<table>
<thead>
<tr>
<th></th>
<th>Prevalent delirium</th>
<th>Incident delirium</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Infective (n = 33)</td>
<td>Non-infective (n = 24)</td>
</tr>
<tr>
<td>Age, years</td>
<td>82.9 ± 5.4</td>
<td>84.1 ± 5.7</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>22 (66.7)</td>
<td>21 (87.5)</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>22.9 ± 4.0</td>
<td>23.4 ± 3.5</td>
</tr>
<tr>
<td>Albumin serum levels (gr/dl)</td>
<td>3.0 ± 0.4</td>
<td>3.0 ± 0.4</td>
</tr>
<tr>
<td>Apache II score (0–77)</td>
<td>11.1 ± 3.6</td>
<td>9.6 ± 3.1</td>
</tr>
<tr>
<td>Charlson comorbidity index</td>
<td>3.6 ± 2.9</td>
<td>2.5 ± 1.8</td>
</tr>
<tr>
<td>Drugs on admission, n</td>
<td>5.9 ± 2.7</td>
<td>5.8 ± 3.4</td>
</tr>
<tr>
<td>mini Mental State Examination (0–30)</td>
<td>16.6 ± 7.4</td>
<td>20.1 ± 12</td>
</tr>
<tr>
<td>Clinical Dementia Rating Scale (1–5)</td>
<td>1.6 ± 1.1</td>
<td>2.0 ± 1.2</td>
</tr>
<tr>
<td>Instrumental activities of daily living (functions lost)</td>
<td>4.9 ± 2.9</td>
<td>5.6 ± 2.7</td>
</tr>
<tr>
<td>Barthel Index on admission</td>
<td>33.7 ± 23.5</td>
<td>33.7 ± 24.2</td>
</tr>
<tr>
<td>C-reactive protein on admission*</td>
<td>4.5 ± 5.0</td>
<td>2.7 ± 3.3</td>
</tr>
<tr>
<td>at delirium onset</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>at delirium resolution</td>
<td>2.0 ± 2.4</td>
<td>0.9 ± 0.9</td>
</tr>
<tr>
<td>Delirium duration (days)</td>
<td>11.7 ± 8.6</td>
<td>12.0 ± 6.7</td>
</tr>
</tbody>
</table>


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**SALVATORE SPECIALE**

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Letters to the Editor

Our data show an association between high C-RP level on admission and both prevalent and incident delirium, but it remains a non-specific marker for a condition that is notoriously heterogeneous in its aetiology and presentation. We were unable to replicate the suggestion by Macdonald et al. that low initial C-RP level predicts recovery nor did it predict delirium duration or severity. Greater patient heterogeneity in our larger study of unselected patients may account for C-RP being less discriminatory in this population.

Multiple pathologies are implicated in the causation of delirium. The focus on a single pathway or marker risks over-simplifying the condition, which is potentially misleading clinically. Nevertheless, quantification of the delirium syndrome remains a challenge and, as suggested by George and Mukaetova-Ladinska [4], even non-specific markers can be helpful in raising awareness of this most frequent complication of hospitalisation for older people.

Conflicts of interest

None

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Reply

We thank Speciale and his colleagues, and White and her colleagues for their comments on our paper, and for their data which confirms an association between C-RP and delirium. We accept, of course, that differences between our studies of the patient group (particularly those with and without inflammatory causes of delirium), prevalent and incident delirium rates, methods of data analysis, and in other factors will explain differences in findings. In more recent works (submitted), we suggest that C-RP levels themselves may not be as relevant in delirium as the cytokines and other markers. White and colleagues’ caution against the risks of oversimplification is well taken; however, examining medical students at the end of their geriatric placements using a video of an obviously delirious patient suggests that if we oversimplify delirium by equating it with C-RP levels, at least for this audience and their trainers, we will be doing less harm and more good than at present.

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