Television viewing and asthma: spurious relationship?

In the April 2009 issue of Thorax, Sherriff and co-authors report on data taken from the ALSPAC study, addressing the association between television viewing in early childhood and the development of asthma.1 They found that, after adjustment for body mass index, there was a relationship between the two, showing a significant trend. I was surprised to see that television viewing was viewed solely as a proxy for a sedentary lifestyle, but not as being associated with other risk factors for developing asthma. For example, although the authors corrected for smoking during pregnancy, they did not include parental smoking at home in their model. It is not unlikely that among parents of children who were reported to have been watching television for longer, many of them were smoking in the presence of their child.

Adjustment for such additional factors is warranted before discussing the consequences of the study findings.

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Author’s reply

We thank Dr van der Wouden for his interest in our paper.2 He raises the question as to whether our observations could be mediated (confounded) or modified by contemporaneous environmental tobacco smoke (ETS) exposure during periods of TV watching. We did find that parents of children with longer reported TV watching were more likely to report that the child was exposed to tobacco smoke: 17.4% of children watching no TV at all were exposed to postnatal ETS, 25% of children watching less than 1 h per day, 53.1% of children watching 1–2 h per day and 42.3% of children watching 2 h or more per day (p linear <0.001). However, only 6.4% of children exposed to postnatal ETS reported asthma at 11.5 years compared with 5.9% not exposed (p for difference between proportions 0.62).

Therefore, despite the association of ETS exposure with reported TV viewing, the lack of a strong association of ETS with asthma at 11.5 years in children asymptomatic up to 3.5 years made it unlikely that postnatal ETS had an independent effect on asthma development in this sample.

In our paper, we chose to adjust the final model for prenatal tobacco smoke exposure only. This was chosen because there was a high degree of co-linearity between prenatal and postnatal smoking in this population and prenatal exposure has been reported to be more strongly associated with asthma in several studies (see the recent meta-analysis by Pattenden et al.).3 We have previously reported that prenatal exposure is associated with early onset wheezing,4 but that neither prenatal nor postnatal exposure to ETS was associated with later onset or persistent wheezing, more likely to be phenotypes associated with asthma. By excluding children who wheezed at any time before 3.5 years from our study, we think it is likely that we have attenuated any potential effect of early smoke exposure on the outcome. Finally, when we considered reported postnatal ETS as a covariate in our final model along with prenatal exposure, we found no attenuation of the association of TV viewing with asthma.

We also considered the possibility that postnatal ETS may have modified the association of prolonged TV viewing with asthma at 11.5 years, as suggested by the correspondent, but a formal test of interaction between TV viewing and ETS on asthma outcome did not support this (p=0.78). Asthma prevalence at 11.5 years stratified by postnatal ETS exposure is shown in table 1.

Table 1 Asthma prevalence at 11.5 years stratified by postnatal ETS exposure

<table>
<thead>
<tr>
<th>TV viewing</th>
<th>Not exposed</th>
<th>Exposed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
<td>4.3 (3/70)</td>
<td>10 (1/10)</td>
</tr>
<tr>
<td>&lt;1 h per day</td>
<td>4.5 (30/663)</td>
<td>3.2 (6/187)</td>
</tr>
<tr>
<td>1–2 h per day</td>
<td>5.5 (52/952)</td>
<td>5.9 (26/442)</td>
</tr>
<tr>
<td>≥2 h per day</td>
<td>9.1 (39/429)</td>
<td>8.8 (22/249)</td>
</tr>
</tbody>
</table>

ETS, environmental tobacco smoke.

Steroid-induced hyperglycaemia and pulmonary disease

Chakrabarti and colleagues recently reported that hyperglycaemia within 24 h of admission could be used as a predictor of outcome during non-invasive ventilation in uncomplicated chronic obstructive pulmonary disease (COPD).1 Hyperglycaemia was unrelated to prior oral corticosteroid use in this study, but duration of steroid preceding admission was not reported. Furthermore, as this group included only 18 patients it would be insufficiently powered to detect a modest rise in glucose. Doctors are vigilant to the occasional patient who develops symptomatic hyperglycaemia whilst taking steroids, but are less attentive to small changes in glucose. Li et al recorded complications of steroid treatment in a cohort of 1291 patients with SARS (severe acute respiratory
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