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CORRESPONDENCE

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 that 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[1] 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, 33.1% 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 were 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

% (n) with asthma at 11.5 years		
TV viewing	**Not exposed**	**Exposed**
Not at all	4.3 (3/70)	10 (1/10)
<1 h per day	4.5 (30/663)	3.2 (6/187)
1–2 h per day	5.5 (52/952)	5.9 (26/442)
2+ h per day	9.1 (39/429)	8.8 (22/249)

ETS, environmental tobacco smoke.

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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 decompensated 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

syndrome) of whom 1084 (84%) were treated with methylprednisolone while 207 (16%) received no steroid treatment.[2] Glucose levels were the same at baseline in both groups but in those treated with steroid the mean value rose significantly. The highest blood glucose in the methyprednisolone group was 8.68 mmol/l (±4.8) compared with 6.39 mmol/l (±3.71) in the non-steroid cohort ($p<0.05$).[2] This change is comparable with the 1.8 mmol/l increase observed with hydrocortisone in a multicentre randomised trial of steroids in sepsis.[3]

An increase of this magnitude appears trivial, but significantly alters glucose levels within the lung. Airway surface fluid is a key element of pulmonary defence, and glucose is normally maintained 3–20 times lower than plasma levels by active transport mechanisms.[4] The latter has a threshold of 6.7–9.7 mmol/l and glucose increases in airway fluid when plasma levels exceed this value. Furthermore, pulmonary inflammation disrupts epithelial integrity and also leads to a rise in lung glucose. Airway surface fluid contains surfactant proteins A and D, which not only are important host defence molecules against a broad spectrum of pathogens but, in addition, possess a number of immunoregulatory properties. These proteins are members of the collectin family, which recognise carbohydrate moieties on microorganisms through their lectin domain. The latter also binds glucose, which may act as a competitive inhibitor of surfactant proteins.[5] It is little surprise, therefore, that raised airway fluid glucose promotes pulmonary inflammation and infection.[4]

Corticosteroids are an important treatment modality in many pulmonary and extrapulmonary diseases. It is likely that in many diseases such as COPD, interstitial lung disease and asthma, modest hyperglycaemia associated with steroid use abrogrates the beneficial anti-inflammatory effects of these drugs. Further investigation of this phenomenon is warranted not only in COPD, but also in other pulmonary diseases in which steroids are commonly used.

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Authors' response

We thank Dr Wise and colleagues[1] for their thoughtful response to our work in chronic obstructive pulmonary disease patients with decompensated hypercapnic respiratory failure.[2] We believe that modest hyperglycaemia is a useful way of identifying patients at greatest risk of treatment failure with non-invasive ventilation, but we are more cautious than those correspondents in implicating corticosteroid use either acute or chronic as a major aetiological factor. Our study was clearly underpowered to exclude such an association but we did not see any trend towards a worse outcome in relationship to previous oral corticosteroid use. The issues reported in the patients with severe acute respiratory syndrome taking methylprednisolone are less likely to apply in our patients in whom the dose of systemic corticosteroids used to treat chronic obstructive pulmonary disease exacerbations is significantly lower than in the severe acute respiratory syndrome study or than that reported in the USA.[3 4] Previous use of inhaled corticosteroids can be associated with clinically diagnosed pneumonia, but hyperglycaemia was not an issue in that large trial nor is pneumonia incidence always increased by inhaled steroid use.[5 6] The mechanisms suggested by which hyperglycaemia promotes lung infection are plausible but will be difficult to test in humans. Disappointingly, recent data suggest that tightly controlling hyperglycaemia in an intensive care unit setting is associated with worse rather than better outcomes, which support our view that this may be a marker of disease severity rather than a causal factor leading to a worse outcome.[7]

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Pneumocystis jirovecii in pleural infection: a nucleic acid amplification study

Pleural infection is associated with 20% mortality in the 80 000 new cases per year in the UK and USA. *Streptococcus* species cause ~50% of community-acquired bacterial pleural infection.[1] *Staphylococcus aureus* and anaerobes are isolated in 8% and 20% of cases, respectively, and 12% of pleural infections yield polymicrobial cultures. However, even using culture and nucleic acid amplification techniques (NAATs), 26% of cases remain microbiologically obscure.

The negative microbiology may be due to previous antibiotic treatment, varying pathogen prevalence in different pleural fluid locules (already known to vary biochemically[2]) or the presence of organisms that are difficult to detect using conventional techniques. One such possible organism is *Pneumocystis jirovecii*, which requires specialist diagnostic techniques (eg, Grocott–Gomori methenamine silver staining or NAATs).

P jirovecii has been identified in sputum and bronchoalveolar lavage (BAL) fluid from both immunocompromised and immunocompetent individuals—it has been isolated from BAL fluid using NAATs in 18% of patients with lung disease without HIV