

## Exposing children to secondhand smoke

In the October issue of *Thorax*, Leonardi-Bee *et al* reviewed the effects of parental smoking on the uptake of smoking by children.<sup>1</sup> They conclude that exposure to smoking within the family is a significant determinant of subsequent smoking.

One area that this study did not address was the effect of the site of the smoking on exposure.

We have studied urinary cotinine levels in children attending an asthma clinic and correlated them with the self-reported smoking habits of their carers. Thirty-six girls and 64 boys aged between 30 and 164 months attending our asthma clinic were recruited if at least one carer smoked. The carers completed a questionnaire about their smoking habits and the child's urine was tested for cotinine using NicAlert, a commercially available reagent strip. Levels of 0–10 ng/ml correspond to minimal or no nicotine exposure, 10–100 ng/ml to moderate to high SHS exposure and >100 ng/ml to active smoking. Ten further children were recruited from the same clinic from non-smoking families and acted as controls.

Seventy-two 'smoking' families (72%) reported only smoking outside the house ('outside' smokers) while 28 parents (28%) did not take any harm reduction strategies ('everywhere' smokers). Three (4%) of the children in outside smoking carers' group had levels indicative of active smoking and were excluded from analysis. Urine testing showed measurable cotinine (10–100 ng/ml) in 27 of 28 (96%) and 61 of 69 (88%) everywhere and outside smokers' children, respectively ( $p=0.20$ ). Only one child in the control group (10%) had detectable cotinine ( $p<0.0001$ , compared with both everywhere and outside smokers' children) and it transpired that she was exposed to SHS at school.

Our findings suggest that there is little or no difference between nicotine exposure in those children of outside smokers and those

in direct contact with smokers (everywhere smokers). Even if a proportion of our outside smokers were in fact smoking indoors, the very high percentage of this group that had raised cotinine levels indicates that most of the 'outdoor' smokers are indeed exposing their children to SHS. This is consistent with reports previously published, showing that nicotine can be transmitted through a number of third hand routes.<sup>2</sup>

Harm reduction strategies (effectively limiting the exposure) are reported to have some benefit in reducing nicotine exposure although our data suggest that reduction may have been overestimated in the past.<sup>3</sup> Using urinary cotinine levels may also be useful in helping advice on smoking cessation.<sup>4</sup> In our study we were able to use the results which were shown to parents at the time of the clinic, and they did seem to focus parental attention on the problem. We found that a number of parents reported cessation of smoking following the test and that their children were free of urinary cotinine when tested.

We suggest that measurement of urinary cotinine can be a useful adjunct when discussing parental smoking and that outside smoking may not protect children from SHS exposure as much as previously thought.

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