18–29 years, and the age groups are unclear for the countries that have not published individual results.[7]

We urge Interphone to fill in the gaps in our Tables 1 and 2, so as to make full comparison with our data possible. Currently, we have presented results on the association of use of wireless phones and malignant brain tumours among deceased cases, that were excluded from our study, using deceased controls. These results confirm our previous findings of an increased risk for malignant brain tumour among mobile phone users.[8]

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doi:10.1093/ije/dyq246
Advance Access publication 17 December 2010

A pilot study to explore whether airborne endotoxins play a role in the association between environmental tobacco smoke and non-respiratory, smoking-related diseases

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Lipopolysaccharides (endotoxins produced by Gram-negative bacteria) are present on the surface of cigarettes and have been implicated in a number of diseases. Active smoking predisposes to periodontal disease which, in turn, facilitates gastrointestinal absorption of endotoxins. Serum endotoxin concentrations are higher among active smokers and are associated with risk of cardiovascular disease.[1] In addition to being present on the surface of cigarettes, endotoxins are present in both mainstream and sidestream cigarette smoke.[2] Inhaled airborne endotoxins have been implicated in the pathogenesis of respiratory disease.[3] In a guinea pig model, endotoxins have been shown to penetrate the lung barrier and be detectable in the blood.[4] Similarly, non-smokers with occupational exposure to organic dust containing high levels of endotoxin have been shown to have increased plasma concentrations of lipopolysaccharide.[5] However, no previous study has examined whether exposure to environmental tobacco smoke is associated with increased serum endotoxin concentrations.

In a pilot study, we compared serum endotoxin levels in three groups of individuals: 10 self-reported never smokers who lived with non-smoking partner and had a serum cotinine concentration of $\leqslant$0.1 ng/ml; 10 self-reported never smokers who lived with partners who smoked and had a cotinine concentration of 6–11 ng/ml; and 10 self-reported current smokers (at least 20 cigarettes per day) who lived with non-smoking partners and had a cotinine level of $\geqslant$600 ng/ml. Cotinine was assayed using gas chromatography with a specific nitrogen phosphorus detector. The lowest level of detection was 0.1 ng/ml. Serum endotoxin was measured using a kinetic turbidometric Limulus amoebocyte lysate (LAL) assay, following heat inactivation (1:10 serum, 15 min at 70°C). Diluted serum samples (0.1 ml) were assayed with 0.1 ml of LAL and incubated at 37°C for 75 min. Optical density readings were obtained every 30 s

at 340 nm and spike recovery was calculated. The limit of detection was 0.05 Endotoxin Unit/ml (EU/ml).

The median serum endotoxin concentrations were 0.487 EU/ml [interquartile range (IQR) 0.050–0.944] among never smokers protected from environmental tobacco smoke, 0.402 EU/ml (IQR 0.050–0.678) among never smokers exposed to environmental tobacco smoke and 0.683 EU/ml (IQR 0.284–7.000) among current smokers. Compared with never smokers, current smokers had significantly higher serum concentrations of endotoxin (Mann–Whitney U-test, $P = 0.006$). There was no statistically significant difference in serum endotoxin concentrations between never smokers protected from environmental tobacco smoke and never smokers exposed to environmental tobacco smoke (Mann–Whitney U-test, $P = 0.436$).

In conclusion, we found no evidence that airborne endotoxin from cigarette smoke can translocate to the blood. Therefore, this mechanism is unlikely to play a role in the association between exposure to environmental tobacco smoke and non-respiratory smoking-related diseases such as cardiovascular disease. Our findings require corroboration in a larger study.

Funding

This study was supported by a grant from NHS Health Scotland.

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doi:10.1093/ije/dyq236
Advance Access publication 10 December 2010