Tobacco

United States Department of Health and Human Services. June 2006
The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General.

The report clearly asserts that lung cancer, heart disease and sudden infant death syndrome are causally related to exposure to environmental tobacco smoke. Asthma is not caused by exposure but there is limited evidence that frequency and intensity can be increased by it.

The report provides information that could be used as a basis for risk assessment. It follows 20 years after the 1986 Surgeon General's report on the same subject. New topics include sudden infant death syndrome (SIDS), non-lung cancer and adult heart disease. The author refers to exposure to environmental tobacco smoke (ETS) as 'secondhand smoking'.
Much of the information on which the new report is based is available at http://www.cdc.gov/tobacco under the heading "Smoking and Health Resource Library". Demographic analysis and relative risks taken from epidemiology studies can be used to calculate the attributable fraction and rates of smoking related disease. Information on latency gives an idea of the timescale over which manifestation can be expected. Exposure to risk is not the same as material contribution.

General conclusions are that second hand smoke is carcinogenic and that it increases the risk of heart disease in adults. In children/infants there is increased risk of SIDS, bronchitis, pneumonia and ear infections and increased risk (frequency and severity) of asthma symptoms.

> *"In 2005, it was estimated that exposure to secondhand smoke kills* (sic) *more than 3,000 adult nonsmokers from lung cancer, approximately 46,000 from coronary heart disease, and an estimated 430 newborns from sudden infant death syndrome."*

In more detail:
In the US, annual attributable deaths

- from lung cancer are between 3,423 and 8,866
- from cardiac-related illnesses are between 22,700 to 69,600

Also, about 202,300 episodes of childhood asthma (new cases and exacerbation episodes), between 150,000 and 300,000 cases of lower respiratory illness in children, and about 789,700 cases of middle ear infections in children occur each year in association with ETS exposure.

Attribution and association do not, by themselves indicate that causation is established; all, or a proportion of, the attributable cases could arise because of confounding i.e. exposure to ETS correlates with another exposure which actually does the harm.

The new report assess whether or not there is sufficient evidence of causation. Causation was assessed by expert reference to evidence from epidemiological studies and from studies related to mechanism of disease action.

The Surgeon General considers the following outcomes to be **causally related to exposure** to ETS:

- Respiratory illness, reduced lung function and slower lung growth in children.
- Coronary heart disease in adults Pooled relative risks from meta-analyses indicate a 25 to 30 percent increase in the risk of coronary heart disease from exposure to ETS. i.e the relative risk is around 1.25.
- Lung cancer in non-smoking adults The pooled evidence indicates a 20 to 30 percent increase in the risk of lung cancer from second hand smoke exposure associated with living with a smoker. i.e the relative risk is around 1.25.
- SIDS pooled risk estimate of the order of 2.0; population attributable risk = 10%
- Recurrent otitis media
- Ever having asthma is causally related to parental smoking

Risk estimates are only really meaningful where causation is certain and where studies allow a pooled relative risk estimate and where there is sufficient detail on population exposures. Risk estimates could be made for 'recurrent otitis media', 'ever having asthma' and for 'respiratory illness' but these are not well defined outcomes in the context of liability exposure and it may not be worth while to pursue these details.

There are indications of **uncertain causation** for the following outcomes:

- Acute respiratory infections in children
- Greater severity of asthma in children

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- Pre-term delivery
- Reduced birth weight
- Childhood leukaemia
- Childhood lymphomas
- Childhood brain tumours
- The onset of childhood asthma
- Breast cancer
- Nasal sinus cancer in non smokers
- Stroke
- Atherosclerosis
- Subjective outcomes:
    - Odour annoyance
    - Nasal irritation
- Biological effects in healthy people:
    - Cough
    - Wheeze
    - Chest tightness
- Biological effects in people with illness:
    - Acute decline in lung function for asthmatics
- Adult onset asthma
- COPD. 85% to 90% of COPD is attributable to active smoking. In non smokers the relative risk due to ETS is of the order 1.2 to 2.0. Around 1 to 5% of non smokers have COPD.

There is no meaningful pooled risk estimate when causation is uncertain. However, the epidemiology can be used to run a sensitivity analysis, providing estimates of the likely ranges of attributable risk. Such analysis is beyond the scope of this report. Many of the outcomes listed above are well-defined enough to give rise to the possibility of liability exposure.

There was **inadequate or no data suggesting causation** of:

- Reduced male fertility or fecundity
- Spontaneous abortion
- Neonatal mortality
- Congenital malformations
- Cognitive functioning in children
- Behavioural problems
- Child growth/development
- Childhood cancer
- Tonsillectomy in children
- Atopy
- Nasopharyngeal carcinoma in non smokers
- Cervical cancer
- Biological effects in healthy people:
    - Acute decline in lung function
    - Accelerated decline in lung function over a lengthy period
- Biological effects in people with illness:
    - Morbidity in persons with COPD.

Control of exposure:
The report finds the following key points in relation to prevention of exposure at work:

- Current heating, ventilating, and air conditioning systems alone cannot control exposure to ETS.
- The operation of a heating, ventilating, and air conditioning system can distribute ETS throughout a building.
- Establishing smoke-free workplaces is the only effective way to ensure that ETS exposure does not occur in the workplace.

Objective analysis of the effects of exposure measures can only really be made by means of biological monitoring. The report concludes that at this time, cotinine, the primary proximate metabolite of nicotine, remains the biomarker of choice for assessing ETS exposure. Measurements of one marker may not wholly reflect an exposure to other components of concern as a result of involuntary smoking.

Comment
Of those outcomes which are regarded as causally related to ETS exposure [heart disease, lung cancer and SIDS] none is at such a rate in the general population that causation could be presumed in any given case. Even among the population that is known to have experienced exposure to ETS the risk is

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much less than 50% (actually around 20%). In our view the contribution made by exposure to ETS is uncertain even in cases where there are no competing environmental causes proposed. It would be beyond the scope of the current evidence to objectively find acceleration or material contribution.

For heart disease and for lung cancer the independent additional risk from ETS exposure is of the order of 25%. Those who regard exposure to risk being equivalent to material contribution would probably regard this as compensable but, so far, legal precedent in the UK would tend to exclude this as a cause of action in its own right.

Claims for damages in relation to lung cancer caused by direct smoking have not succeeded in the UK courts. Smoking has been recognised as aggravating the effects of exposure to asbestos; possibly the only situation where exposure to one agent has been shown to aggravate the effect of exposure to another. In our view, risks that do not interact with each other should be dealt with according to the precedent set in *Wilsher*.

Adult onset asthma is classified as having an uncertain causal relationship with exposure to ETS. This does not preclude the possibility that a judge could find a causal link in a given case but should indicate the possibility of making out a foreseeability defence even if he does.

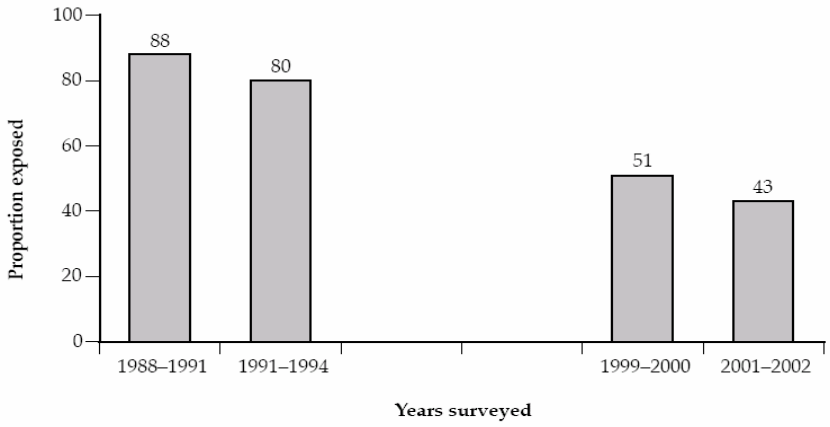
The effect of ETS on those adults with current asthma is also regarded as uncertain.

When combined, these two findings would suggest that it is unlikely that an adult would succeed in any asthma related ETS claim. Any occupational exposure policy that has been justified on a presumption that ETS aggravates asthma would not be regarded as evidence-based [it would be an over-reaction].

There was no or inadequate data suggesting that exposure to ETS increases susceptibility to allergies.

There is a long list of outcomes described as possibly related to ETS exposure. Given that most of these have been studied in depth for many years, the prospects a firm causal link being established seem remote, especially as exposure rates are falling.

Figure 4.1 **Trends in exposure* of nonsmokers† to secondhand smoke in the U.S. population, NHANES‡ 1988–2002**



*Serum cotinine ≥0.05 nanograms per milliliter.
†Aged ≥4 years.
‡NHANES = National Health and Nutrition Examination Survey.
Source: Adapted from Pirkle et al. 2006.

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