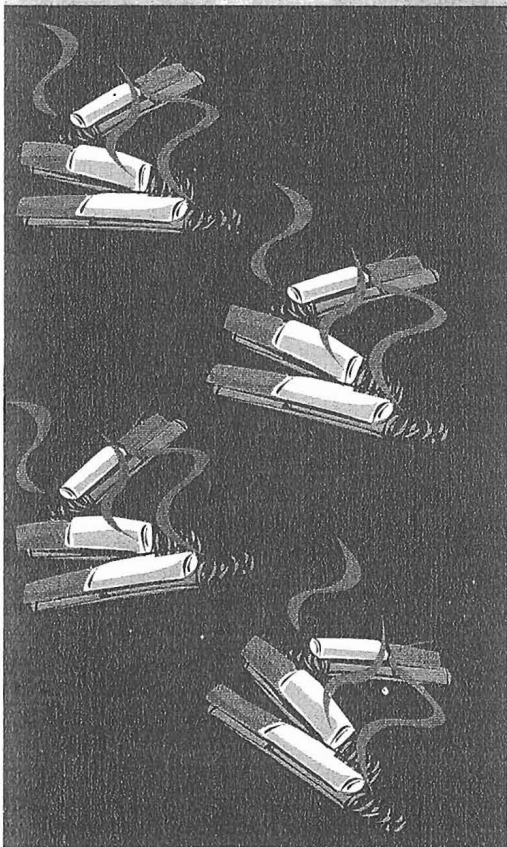


Involuntary Exposure to Tobacco Smoke in Children

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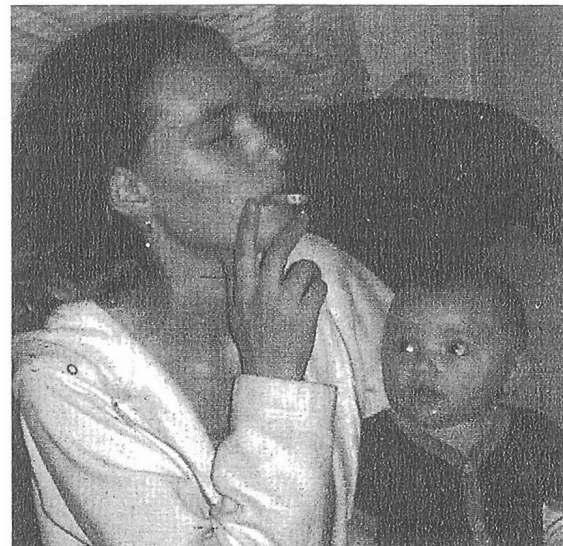


Tobacco smoke contains more than 50 human and/or animal carcinogens and numerous irritant and toxic compounds.¹ Active smoking is known to cause characteristic respiratory, cardiovascular, and carcinogenic adverse events. Smokers inhale both mainstream and side-stream smoke, which are qualitatively similar, although differing somewhat in concentrations of contaminants. Passive smoking results in exposure to side-stream and exhaled mainstream smoke contaminants. The terms environmental tobacco smoke (ETS), second hand smoke (SHS), and involuntary exposure to tobacco smoke have all been used to describe this phenomenon. This year's report of the Surgeon General emphasizes the use of the term "involuntary exposure to tobacco smoke" to emphasize the lack of consent by the exposed non-smoker. Children are particularly disadvantaged when trying to avoid exposure because their consent is rarely sought, they are often in situations where they are unable to leave the contaminated area (e.g., maternal smoking for young children), and they lack the social standing to be given consideration or express preference. Children, because of their relatively higher rates of ventilation relative to body mass, receive significantly higher doses from the same exposure than adults. Consequently, they are at increased risk from SHS, not just because of immature still developing organ systems, but also because of the increased dose. Results of studies of a causal relationship between SHS and adverse human health outcomes are sometimes inconsistent, often a result of differences in study design, e.g., cross-sectional versus longitudinal, use of biomarkers to measure dose, population selection.

Jaakkola² points out the effects of SHS on the child from maternal smoking usually, but not always, exceeds that from paternal smoking. This is thought to be a result of the influence of maternal smoking during pregnancy and to the closeness of the infant or child to the mother after birth resulting in higher exposures to the child. Cotinine freely diffuses through the placenta, resulting in significant prenatal fetal exposure. Detectable abnormalities of lung function have been demonstrated soon after birth in infants of smoking mothers, such as decreased airway compliance and increased airway hyperresponsiveness. The causal strength of the effects of maternal SHS exposure on the development of fetal lung tissue and growth of the newborn are weaker and more often inconclusive.

Demography

The home is the primary source of SHS exposure for young children. The family car may also be a source of significant exposure. A child's exposure profile to tobacco smoke may vary considerably during different periods of his or her life, and exposures back to pregnancy or early childhood should be considered



in determining risk and causation. Children, particularly young children who spend most of their time in the home with their mothers, are at increased risk for even greater exposures to tobacco smoke if their mother smokes. Forty to sixty percent of children in the United Kingdom and approximately 43% of children in the United States are exposed to ETS at home.³ In England, childhood exposure, measured by cotinine levels, has fallen by 50% since the 1980s. In the United States urinary cotinine levels in children and adolescents have fallen approximately 70% in the past decade.¹

Respiratory Effects

Two of the major challenges in differentiating the pre-natal from post-natal effects of SHS have been 1) the difficulty in finding smoking mothers who did not smoke during their pregnancy but resumed after delivery and 2) the long latency periods of many of the effects of SHS exposure. Although inter-study differences exist in the respiratory health outcomes evaluated, in general, the end-points have been some combinations of wheezing (acute and chronic), cough, phlegm, diagnosis of asthma, breathlessness, and hospital admission or the requirement for urgent care. In a series entitled "Health effects of passive smoking" [in children] in the journal *Thorax* from 1997-1999, Stachan and Cook reviewed the literature.⁴⁻¹¹ A meta-analysis by Strachan & Cook of 38 studies in infancy and early childhood (years 0-3) reported a pooled odds ratio (OR) of 1.42 (either parent smoking) and 1.72 (maternal smoking) for the increased risk of acute lower respiratory illness from SHS exposure, concluding this was a casual association. This effect was also present in families where the mother was not the smoker in the home. The finding of paternal involvement as a factor indicates not

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only the likelihood of a significant effect from paternal smoking alone, but also that this effect is at least due to some contribution from post-natal SHS exposure.¹¹ Evidence for a causal effect persisted into school age. While pooled ORs for this age group were somewhat lower, the strength of the association remained “very likely to be causal” for asthma, wheeze, cough, phlegm, and breathlessness. Furthermore, a dose-response relationship was evident.⁶

Investigating the occurrence of wheezing illness from 1-6 years of age, Strachan and Cook found maternal smoking resulted in a pooled OR = 1.31 up to age 6, but only 1.13 subsequently. This adverse effect appeared to occur mainly as non-atopic “wheezy bronchitis.” However, for *diagnosed* asthmatics parental smoking was associated with much more severe disease, suggesting SHS is a co-factor triggering acute wheezing, but not a cause of asthma.⁸ Consistent with this theory, SHS has not been shown to increase the risk of bronchial hyperresponsiveness, although limited evidence shows increased variability in peak expiratory flow rates in SHS-exposed children.⁵ In a report published in 2005, a scientific review panel on toxic air contaminants convened by the California Environmental Protection Agency (Cal/EPA) concluded parental smoking, both prenatal and postnatal, not only caused asthma exacerbations in children, but also caused induction of asthma in

children *and adults*. Moreover, they reported a causal relationship between parental smoking and exacerbation of disease in children with cystic fibrosis is likely.¹² Maternal smoking is associated with a small but statistically significant reduction in lung function as reflected in changes in FEV1, and mid-expiratory and end-expiratory flow rates in school aged children. Four of six longitudinal studies in children demonstrated an adverse effect of SHS on lung function as measured by FEV1 and, depending on the study, FEF 25-75% or mid-expiratory flow (MEF). Maternal smoking has been a stronger determinant of lung function than paternal smoking in most studies, with the strongest effect seen from maternal smoking during either pregnancy *or* infancy.² These effects are also evident at birth or in the neonatal period in some studies of neonates, suggesting pre-natal SHS exposure, in addition to exposure after birth, can have a negative impact on lung growth.⁷

Although parental smoking does not appear to increase the risk of the development of allergy in exposed children,⁹ SHS significantly increases the risk of developing acute and chronic middle ear disease.¹⁰ However, the data for tonsillectomy and/or adenoidectomy is conflicting—four studies demonstrating increased risk and one study showing no difference.¹⁰

Lower respiratory tract illnesses are common in children, with lower respiratory tract infections (acute bronchitis, bronchiolitis, pneumonia, respiratory syncytial virus), wheezing and cough occurring often in infants and young children.² Studies have consistently demon-

strated an increased risk of lower respiratory tract illnesses beginning early in life in young children exposed to SHS. One meta-analysis found a summary odds ratio of 1.57 for either parent smoking and 1.72 for maternal smoking. Studies in the United Kingdom and New Zealand looking at pneumonia and bronchitis found adjusted odds ratios of 1.96 and 1.56, respectively, for either parent smoking and 2.79 and 1.83, respectively, for both parents smoking. A study in the People’s Republic of China also suggested a dose-response relationship for SHS and the development of pneumonia or bronchitis in infants and very young children. Two different meta-analyses found evidence for increased odds ratios for the development of acute otitis media in children exposed to SHS. Strachan and Cook reported the risk for chronic middle ear disease is greater than for acute disease.¹⁰

Non-Respiratory Effects

Two recent reviews of the health effects of SHS in children have discussed the evidence for causation of various non-respiratory adverse outcomes.^{3, 13} In addition, Cal/EPA and the U.S. Surgeon General recently released comprehensive reports regarding the adverse health outcomes of SHS.^{1, 12} In addition to respiratory effects, these reviews include discussion of the evidence for causation of adverse health outcomes in the prenatal and perinatal periods, infections, oncogenesis, and neurodevelopment. Although the reviews are not consistent in all of their conclusions, most are concordant regarding cause and effect—either as “sufficient to support a causal relationship” or “suggestive but not sufficient.”

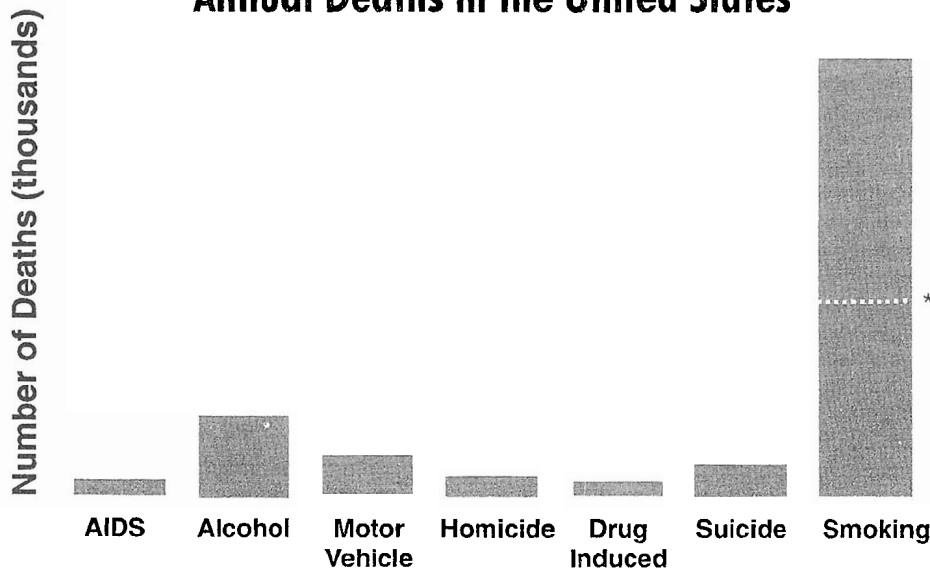
Prenatal and perinatal periods

Adverse effects induced by prenatal and perinatal maternal smoking on the fetus are well documented. It is not surprising that some of these may occur as a result of maternal SHS exposure. SHS is associated with an increased risk of preterm birth. Additionally, low birth weight or a small reduction in birth weight appears to be associated with prenatal SHS exposure. One meta-analysis found a pooled birth weight difference of -28.5g. Genetic polymorphisms in pregnant smokers may cause significantly greater reductions in birth weight. Maternal smoking during pregnancy also increases the risk of group B streptococcus colonization.

Infancy and childhood

The risk of sudden infant death syndrome increases two-fold for infants with mothers who smoked during pregnancy or in the post-natal period. The results for paternal smoking only is inconsistent, with some studies showing increased risk and others not. The increased risk of upper and lower respiratory tract infection in a dose-dependent manner was discussed earlier.

Comparative Causes of Annual Deaths in the United States



Source: CDC

* Also suffer from mental illness and/or substance abuse

Compounds in Tobacco Smoke

An estimated 4,800 compounds in tobacco smoke



Gases

- Carbon monoxide
- Hydrogen cyanide
- Ammonia
- Benzene
- Formaldehyde

Particles

- Nicotine
- Nitrosamines
- Lead
- Cadmium
- Polonium-210

11 proven human carcinogens

SHS exposure is also a risk factor for the development of pulmonary tuberculosis immediately following infection and for invasive meningococcal disease. Although data exist supporting a causal role for SHS in the development of cognitive and behavioral problems, they are inadequate to infer any causal relationship. The evidence is suggestive but not sufficient for a causal relationship with childhood leukemias, lymphomas, and brain tumors.

The clarification of possible causal relationships between SHS and adverse health outcomes continues as new data from better designed studies become available. More longitudinal studies, investigation of genetic differences for susceptible populations, identifying critical periods, and the focus on higher dose exposures (more likely to show stronger associations) will continue to teach us more about an already existing surfeit of established adverse effects. Some of the difficulties inherent with this type of investigation include 1) finding children exposed only in utero to SHS (maternal smokers who stop after delivery) and those exposed only after birth (maternal smokers who cease smoking completely during their pregnancy); 2) the lack of the use of biomarkers currently available (cotinine) to more accurately determine dose; and, 3) the challenge of studying potential adverse health effects with very long latency periods, e.g., chronic obstructive pulmonary disease, cardiovascular disease. The most effective way to reduce SHS-related adverse health outcomes is to eliminate SHS exposure. Fortunately, the past decade has demonstrated that the momentum currently exists, at least in industrialized countries, for reducing SHS exposure. Hopefully, this momentum, championed by increasingly widespread public policies eliminating smoking in public areas will translate into improved health for the young adults of tomorrow.

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