Smoking

Defining residential tobacco home policies: a behavioural and cultural perspective

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Commentary on the paper by Spencer *et al* (see page 670)

Passive smoke exposure (PSE) is carcinogenic, linked to cardiovascular and respiratory diseases, increased risk for sudden infant death, and increased severity of asthma, and is generally harmful.[1 2]

According to the behavioural ecological model (BEM), smoking and passive smoke exposure are behaviours influenced by interacting physiological, environmental, and cultural contingencies.[3] Social models, criticism, and praise serve as powerful reinforcing contingencies of lifestyle practices. These interact prominently with physiological and community based contingencies. For instance, once an individual is prompted by the industry to start smoking, nicotine addiction adds physiological consequences for smoking (for example, increased alertness) and for not smoking (for example, increased anxiety). These interact with social contingencies promoted by the industry, media, and social reinforcement from members of personal networks to strengthen the addiction. The strength of the addiction is dependent on the biological addiction to nicotine and the density of reinforcement from social networks. Fortunately, other social networks include people who oppose tobacco smoking, and provide reinforcement for avoiding tobacco, possibly countering the industry influences. These include culture-wide sanctions.

Culture-wide ''values'' define social contingencies that may delimit smoking. One of the more prominent is protecting infants and children from harm, especially if suffering from disease (for example, asthma). To the extent that PSE is viewed as harmful, the community is likely to criticise parents who allow their children to be exposed, especially if very young, ill, or in their own home.

At the legislative level, community policies and related policing and penalty systems can contribute to both direct change in tobacco use and community-wide social reactions to tobacco use and child exposure. Community policies restricting PSE in public buildings, and increasingly in outdoor public places, will reduce smoking and PSE in these environments, but it also may reduce smoking and PSE in private residences.[4] Public building polices may also prompt non-smokers to criticise smokers and to ask them to stop or move from the area. This change in reactions to smoking may generalise to other settings, including private homes, and to the extent that it does, it becomes another cultural contingency impacting smokers' behaviour. Thus, families may be susceptible to social contingencies to delimit their children's PSE, as the larger society adopts cultural standards prohibiting PSE.

One means of protecting children from PSE is the establishment of ''policies'' restricting smoking in the home. These can be created by parents or they may eventually be created by the larger society. The study by Spencer and colleagues[5] in this issue extends the literature on PSE exposure based on harm reduction concepts. It shows that children show lower cotinine levels for families who have ''no-smoking policies'' which restrict all smoking from their home. This strengthens the case for protecting children in their home by promoting residential bans or polices disallowing all cigarette smoking in the home.

However, unlike policies for public buildings, parent residential policies are not enforced by police, employers, building owners, or government agencies. Parents must remove ashtrays, set up signs, and most importantly ask family members and visitors to not smoke or go outside. Coaching interventions show promise for assisting parents in reducing their children's PSE, but these procedures have not yet emphasised formal residential policies.[6] The skills and social contingencies operating for individual mothers or fathers to effect these assertive practices are not captured in the concept of ''home policies''. In order to advance the field of PSE control, the specific assertive practices and the conditions that influence them must be identified and engineered to support parents' establishment of such policies. For instance, can a mother restrict the child's grandmother from smoking in the home; can she do so if the grandmother owns the home? Can she do so, if too poor to move to another residence? Additional research is needed to answer these questions and inform efficacious means of promoting home policies and the behaviour that defines them. Such research is urgent. The ill health effects warrant aggressive efforts to reduce PSE in homes.

As the damage due to PSE has become more evident, agencies that protect the public, such as the judicial system, have begun to delimit PSE for children from parents who are divorcing.[7] As this precedent increases, it will promote other agencies to consider the effects of PSE. The logical extension will be Child Protective Services for neglect or abuse. These institutional interventions deliver severe penalties, such as potential loss of custody of a child. Since the smoking parents, grandparents, and friends are themselves addicted victims of the industry, the use of such severe penalties and their initial selective use in divorce cases or in low income and racial/ethnic minority families, raises risk of prejudicial penalties, making these families a more severe victim of the tobacco industry. This is a questionable use of aversive consequences to alter parenting practices.[8] To offset these relatively draconian penalties, it is vital that the assertive practices necessary to eliminate tobacco from residences be promoted based on empirical evidence of efficacious interventions that emphasise positively reinforcing contingencies, even if the parents do not quit smoking.

In any case, the courts assignment of custody based, in part, on PSE is already influencing parents' smoking and adoption of residential policies. As court penalties become more common and more publicised, they will fuel and justify social sanctions from the public for child PSE. Thus, a cumulative cascade of contingencies is already evolving and how these will compete with the aggressive counter media and counter lobbying of the tobacco industry remains to be seen. It also remains to be seen how public health research can insert more positive means of establishing residential bans in homes to protect children and all family members.

Since most of the ill effects from PSE come from cumulative exposure of even very low doses (for example, <1.0 ng/ml of urine cotinine), and since effects include serious illness, disability, and early death, the social evolution of penalties for child PSE may be the

natural and required early process of curtailing tobacco use, PSE, and the industry that engineers both. This is even more profoundly true when epidemiological studies show that remarkably low doses (for example, less than one part per million) of known toxins, such as benzene, can disrupt progenitor cell function.[9] Since benzene is only one of thousands of such toxins in PSE, this supports the physiological causal path to illness and death. It also accelerates both professionals' and lay audiences' conviction that PSE is too harmful to allow, even if in incredibly small doses.

However, behavioural research must provide parents with the skills and reinforcement to effect change in their homes to protect their children. Behavioural science must also inform community-wide policies that will support parents' efforts to reduce PSE in their homes, without requiring severe penalties. Otherwise, the harm produced by the tobacco industry will extend to the trauma that parents will experience at the loss of child custody.

The Spencer *et al* study provides relatively strong evidence of the value of residential policies restricting all tobacco to outside the home. This sets the stage for determining how to equip parents with the skill and ability to do so, without incurring severe penalties. Movement in this direction will also inform a broader restriction of the tobacco industry.

PSE is completely preventable by elimination of the tobacco industry. Community policies that use positive means of promoting parents to adopt home policies restricting tobacco smoke in the home will contribute to the prevention of children's and others' ill health. This may also be a critical step towards generating a culture that is both anti-tobacco and anti-tobacco industry, creating a public that would lobby for complete elimination of the industry. In the meantime, research must be directed to incremental reduction in PSE for children and all family members, and doing so might lead to the ultimate preventive policy.

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Pollution

Gases from fossil fuel combustion: a danger to infants?

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Commentary on the paper by Klonoff-Cohen *et al* (see page 750)

The combustion of fossil fuels generates a complex mixture of gases, particles and chemicals, many of which have the potential to impair human health.[1] In older adults, epidemiological studies have consistently shown increased cardiovascular mortality associated with increased levels of air pollution.[2] There is also concern, acknowledged by regulatory authorities, that very young children represent another vulnerable population. Many of the factors that could increase the vulnerability of young children to air pollution remain speculative. One known variable is that infants have a higher minute ventilation relative to lung surface area.[3] Thus for the same pollutant concentration, infants' airways will receive a higher exposure than adults. However, paediatric mortality associated with air pollution has not, until recently, been regarded as a major issue. The paper by Klonoff-Cohen and colleagues[4] in this issue is therefore of particular interest. In this case-control study the authors found that monthly sudden infant death syndrome (SIDS) counts tracked with monthly averaged outdoor nitrogen dioxide (NO_2) concentrations, and that high levels of NO_2 over the preceding 24 hours was a significant risk factor for SIDS. Effects were also observed for carbon monoxide (CO), but these were less consistent.

NO_2 is not the most potent gaseous oxidant, and causes less airway inflammation than ozone.[5] Recent research has therefore focused primarily on other pollutants. However, all combustion processes in air directly produce oxides of nitrogen (for example, NO_2 and NO_x). NO_2 is also formed when nitrogen oxide (NO), emitted from vehicle exhausts, reacts with atmospheric ozone. Thus winter NO_2 peaks are associated with low wind speeds and temperature inversions, whereas summer NO_2 peaks are associated with ozone peaks during hot sunny days. In the UK, half of NO_2 emissions are from road transport, and emissions have fallen from 2744 kt in 1990 to 1728 kt in 2000. Widespread exceedences of the 40 $\mu g/m^3$ annual mean limit remain, and are projected to continue over the next decade.[6] A causal relation between NO_2 and SIDS would therefore be an important stimulus for NO_2 reduction strategies. However, as Klonoff-Cohen and colleagues[4] acknowledge, there are some important limitations to their data. First, individual exposure was at best approximate, with concentrations in some cases extrapolated for monitoring stations several kilometres from the home. Nerriere and colleagues[7] compared