Reducing Secondhand Smoke Exposure Among Children and Adolescents: Emerging Issues for Intervening with Medically At-Risk Youth*

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Objective To summarize information on rates of secondhand smoke (SHS) exposure among healthy and medically at-risk pediatric populations, discusses the clinical manifestations of pediatric disease that are exacerbated by exposure, and provide an overview of promising strategies for reducing SHS in vulnerable pediatric populations. **Methods** The success of exposure reduction and smoking cessation interventions implemented with parents of healthy children and those with respiratory disease, in the context of their child's health care, is reviewed. **Results** Concurrent implementation of multiple levels of intervention, including clinical interventions within the medical setting, will help to maximize the reduction in childhood SHS exposure. **Conclusion** Ongoing intervention research and identification of strategies to capitalize on opportunities for providing effective SHS counseling in primary care and specialty clinics will be critical for effective tobacco control among medically at-risk children.

Key words chronic illness; environmental tobacco smoke (ETS) exposure; pediatrics; secondhand smoke; tobacco control.

Secondhand smoke (SHS) is a major preventable cause of morbidity and mortality in the US and children who are exposed to this environmental hazard are particularly at-risk for adverse health outcomes. The US Environmental Protection Agency (EPA, 1992) has classified tobacco smoke as a known human carcinogen. Yet, from 28% to over 40% of children in the US live in homes with a smoker (Pirkle et al., 1996; Schuster, Frank, & Pham, 2002). Nationwide, approximately 15 million children and adolescents are exposed to SHS with the majority of this exposure resulting from parental smoking at home (American Legacy Foundation, 2005; US Centers for Disease Control and Prevention [CDC], 1997).

The purpose of this article is 3-fold. Our first goal was to examine estimated exposure rates among medically vulnerable youngsters who are potentially at-risk for adverse exposure-related health outcomes. Related to this objective was the identification of specific disease and/or treatment-related complications that could be exacerbated by exposure to tobacco smoke in selected vulnerable pediatric populations such as asthma, cancer, sickle cell disease, and cystic fibrosis. Our second goal was to describe SHS reduction interventions that have focused on altering parent smoking behaviors in ways that might decrease their child's exposure without the expectation to quit smoking, as well as those that targeted parent cessation exclusively. Studies that were included for review were identified by computer search (Medline, PsychInfo) and from the authors' research. Review and empirical articles concerning reduction of residential SHS exposure in healthy children and those with respiratory disease from birth through adolescence were included. Only interventions that targeted family or household members were selected as these are typically the primary

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sources of exposure. Stand-alone studies from 1994 to present were identified in order to include some of the earlier hallmark studies in SHS reduction, although studies published prior to 1994 were captured in the review articles presented in this article.

Although all children, whether medically at-risk or not, should be protected from SHS, there are a number of issues that distinguish clinical and research efforts with at-risk populations from those children who are not at risk because of their medical status. The life threatening nature of some childhood diseases and the daily disruptions that accompany sometimes invasive and intensive treatment regimens present a demanding challenge for parents of medically compromised children. Intervention approaches that require parents to change their smoking behaviors while simultaneously attempting to manage stressful aspects of their child's treatment should consider these unique demands. In light of these factors, our third goal was to identify the significant emerging issues for future research in medically at-risk populations based on a synthesis of collective studies to date.

Estimated Exposure Rates Among Medically At-Risk Children

Limited data are available regarding exposure rates among medically at-risk children. There are no national figures regarding the number of children with chronic health conditions who live in households with smokers. Estimates vary, based on smaller studies targeting individual diagnostic groups, yet suggest extensive residential exposure to medically at-risk children. More disturbing is that sick children may be physically unable to remove themselves from the smoke and avoid exposure. Children with chronic medical conditions are likely to spend a significant proportion of their time indoors with their parent or caregiver due to the restrictions of their disease, thereby increasing their risk for SHS exposure if their parent or caretaker smokes.

Results from a large-scale study of children with asthma from urban communities indicate that 39% of families had a caretaker who smoked and 59% of families reported at least one smoker in the home (Kattan et al., 1997). Similarly, Huss et al. (1994) found that 56% of urban minority families with a child with asthma included a smoker. One study that examined exposure among 52 children with sickle cell disease (SCD), aged 2–18 years, found that 42% were exposed to SHS. Patients were considered exposed if either the parent or primary caretaker identified anyone living in the home who smoked any tobacco products in the preceding 2 years (West et al., 2003). Verma, Clough, McKenna, Dodd, and Webb (2000) found that 76% of adult patients with cystic fibrosis (CF) reported current or past childhood exposure to a smoking environment (i.e., exposed to any smokers in their home environment). The incidence of parent smoking has also been documented to be higher in parents of children with CF than in the normal population (Butz & Rosenstein, 1992), suggesting greater risk of harm if smoking occurs in the presence of the child.

In one of two studies to date focused on children with cancer, Tyc et al. (2004b) reported that of 303 consecutive new admissions to a large pediatric oncology institution during a 12-month period, 45% of newly diagnosed patients lived in homes with at least one parent smoker. In 10 additional households in this sample, other family members were the identified smokers. In a smaller study, 47 smoking parents of children undergoing treatment for cancer reported that about 72% smoked in the presence of their child and nearly 70% of parents permitted others to smoke around their child. Almost 58% of parents reported smoking inside the home and close to 77% smoked inside their cars (Tyc, Klosky, Throckmorton-Belzer, Lensing, & Rai, 2004a). Taken together, these results suggest that children with cancer are at-risk for being exposed to SHS throughout their treatment, from multiple sources and in numerous settings. A significant proportion of them are directly exposed to their parents' cigarettes on a regular basis.

SHS-related Health Outcomes

According to the most recent U.S. Surgeon General's report (USDHHS, 2006), there is no risk-free level of exposure to SHS. Exposure to SHS increases children's risk for respiratory illness, ear infections, bronchitis, pneumonia, and reduced pulmonary function (CDC, 1997; EPA, 1992; Etzel, 1994). Declines in lung function from SHS exposure may predispose the development of chronic lung disease in adulthood (Dunn & Zeise, 1997). Children whose parents smoke have a disproportionate number of these and similar medical conditions and their incidence increases with higher levels of exposure (DiFranza & Lew, 1996). Recent reported findings of lower levels of serum Vitamin C in SHS-exposed children provide preliminary evidence of the direct adverse metabolic consequences of SHS (Strauss, 2001). An association between tobacco smoke exposure and metabolic

syndrome in adolescents has also recently been demonstrated (Weitzman et al., 2005).

Medically at-risk pediatric populations are at an elevated risk for the damaging health effects of SHS. Of all the childhood diseases for which SHS is a dangerous environmental irritant, asthma has been the most widely studied. The adverse consequences of SHS exposure on the health of children with asthma have been well documented. SHS exposure in childhood has been reported to be associated with the early development of asthma (Joad, 2000), and more severe asthma symptoms (Beeber, 1996; EPA, 1992) that lead to a higher frequency of emergency room visits and hospitalization rates (Beeber, 1996), increased risk of intubation (LeSon & Gershwin, 1995), increased medication usage, and longer recovery periods following hospitalization (Albulhson, Morray, Llewllyn, & Redding, 1997). SHS exposure is estimated to exacerbate the symptoms of ~20% of children with asthma in the US and is a major aggravating factor for 10% of those children (EPA, 1992).

The exact pathologic mechanisms that link SHS exposure to asthma symptoms or severity have not yet been determined. Bronchial reactivity and allergic sensitization have been postulated as two of these mechanisms (Dubois, Oddoze, Badier, Guillot, & Bruguerolle, 1998; Oddoze et al., 1999; Studnicka et al., 1998). Studies have linked SHS exposure and increased airway reactivity in children with asthma (Dubois et al., 1998; Oddoze et al., 1999) and a dose–response relationship among nonasthmatic children exposed to maternal smoke (Studnicka et al., 1998) has also been reported. Additionally, exposure to high levels of maternal cigarette smoking has been associated with increased inner airway wall thickness in infants who died from sudden infant death syndrome (Elliott, Vullermin, & Robinson, 1998). This evidence, although not definitive, suggests another mechanism that may link childhood airway disease to SHS exposure.

Unlike research in pediatric asthma, the relationship between SHS exposure and childhood cancer-treatment-related morbidity has not been extensively investigated. Despite the lack of empirical data to support this relationship, the pediatric oncology community clearly recognizes the deleterious effects of SHS in young cancer patients (Tyc, Hudson, Hadley, & Throckmorton-Belzer, 2001). Children with cancer are at particular risk for adverse health effects secondary to treatment-related toxicities that may be exacerbated by SHS exposure. Acute complications of SHS exposure may include an increased risk for respiratory infections, especially among immunocompromised children. Long-term exposure to high levels of SHS may increase the risk of treatment-related cardiovascular and pulmonary disease (EPA, 1992). Children with cancer are already at-risk for developing second cancers because of treatment-induced and genetic predispositions (Meisler, 1993; Robison & Mertens, 1993) and exposure to SHS may magnify these vulnerabilities.

Evidence of a direct causal relationship between SHS exposure and childhood cancer is slim and a review of 50 published studies has not supported a strong association between in utero and early childhood exposure to parental smoking and the incidence of childhood cancer (Sasco & Vainio, 1999). Other research has suggested that susceptibility to childhood acute lymphoblastic leukemia may be associated with functional polymorphisms in genes encoding carcinogen-metabolizing enzymes that are consequently related to in utero and postnatal environmental exposures including tobacco smoke (Krajinovic, Richer, Sinnett, Labuda, & Sinnett, 2000). Tobacco smoke has also been shown to suppress immunologic function and experimental models have demonstrated abnormalities in cell-mediated immune functions in mouse progeny, following in utero exposure to benzopyrene, a carcinogen in tobacco smoke, that are associated with a high incidence of tumors (Rodriguez et al., 1999).

Limited evidence suggests that the clinical manifestations of other pediatric medical conditions are likely to be exacerbated by exposure to SHS. One study showed that children and adolescents with sickle cell disease who were exposed to SHS at home, had 1.9 times the risk of acute sickle cell crises that required hospitalization than those who lived in nonsmoking households and were not exposed (West et al., 2003). Although the exact mechanism by which tobacco smoke contributes to the development of sickle cell disease and related pulmonary problems is speculative at best, one possibility is that the carbon monoxide in tobacco smoke displaces the oxygen from hemoglobin-binding sites, resulting in reduced oxygen-carrying capacity of red blood cells and tissue hypoxia (Aranow, 1978). Other components of tobacco smoke are also known to injure vascular endothelium, increasing inflammation, and a host of vaso-occlusive effects (Kimura, Nishinaga, Ozawa, & Shimada, 1994).

Among youngsters with cystic fibrosis (CF), parental smoking has been associated with compromised pulmonary function and increased chest infections (Gilljam, Stenlund, Ericsson-Hollsing, & Strandvik, 1990). Children with CF are susceptible to respiratory airway infections and obstruction secondary to abnormal mucus

production (Wood, Boat, & Doershuk, 1976) and tobacco smoke can exacerbate these disease processes. Although the process is not well understood, reduced nasal and sinus mucociliary clearance has been reported in tobacco-smoke exposed individuals, secondary to diminished nasal ciliary beat frequency (Agius, Smallman, & Pahor, 1998), which can contribute to worsened respiratory status in youngsters with CF.

The relationship between exposure and symptom presentation by children with other diagnoses who may be similarly at-risk, such as diabetes, has not been examined. The magnitude of SHS-related morbidity is likely to be substantial in these at-risk children such that interventions to reduce their exposure to SHS are critical. While the face validity for exposure to SHS to compromise the health of children already suffering from acute or chronic disease (and related treatments) is high, formal research is needed to determine the exact level of increased risk, and the features or topography of exposure that conveys the greatest added risks. Objective evidence of the dose response and varying patterns of exposure that might convey important additional risk will inform prevention programs, sensitize providers to query exposure patterns and more aggressively advise parents and caretakers to protect their children from SHS exposure. Such data will also drive policies to more fully protect children in general and health compromised children in particular.

SHS Reduction and Cessation Interventions

SHS Reduction Studies

Efforts to reduce children's SHS exposure, by altering parental smoking behaviors around their child, have been aimed at parents of exposed healthy infants and children with respiratory disease, particularly asthma. Collective results from these trials indicate that they have been successful in significantly decreasing children's SHS exposure, as measured by parent reported number of cigarettes to which the child was exposed (Greenberg et al., 1994; Hovell et al., 1994, 2000a; Wahlgren, Hovell, Meltzer, Hofstetter, & Zakarian, 1997), nicotine dosimeters (Emmons et al., 2001; Hovell et al., 1994; Wahlgren et al., 1997), or urine cotinine (a metabolite of nicotine) assays (Greenberg et al., 1994; Hovell et al., 1994, 2000a, 2002a). Maintenance of effects has been demonstrated up to 2-years post-intervention (Wahlgren et al., 1997). Although not focused on cessation, SHS interventions have indirectly resulted in some parents reducing or quitting smoking (Hovell et al., 1994, 2002a; McIntosh, Clark, & Howatt, 1994). Significant decreases in reported symptom severity and improved health outcomes as well as decreased health care utilization have also been obtained for children with asthma whose parents participated in SHS counseling (Wahlgren et al., 1997; Wilson et al., 2001).

A critical review of 19 SHS exposure reduction studies published between 1987 and 2002, reported small (minimal contact interventions) to moderate (multiple in home counseling sessions) effect sizes for interventions that ranged from minimal contact, physician-based office advice to home-based counseling (Gehrman & Hovell, 2003). These interventions targeted family or household members of healthy infants and children as well as those with asthma. Although based on a small number of studies, interventions in healthy samples appeared to be as efficacious as those conducted among children with asthma in reducing SHS exposure. The most effective interventions included in-person or telephone-based counseling, shaping, modeling, education, and contingency contracting. One-time brief clinical SHS exposure reduction interventions appear marginally efficacious at best. However, more studies with large patient samples providing sufficient statistical power are needed to fully test minimal interventions. Multisession interventions of increased intensity and duration, which involve counseling processes based on learning theory to shape gradual changes in SHS exposure, have yielded the most promising results.

Most SHS trials have provided only limited repeated counseling over 1 –6 months. Despite statistically significant reductions in exposure following repeated counseling sessions, an important proportion of children remain exposed by the end of these trials, when monitoring typically occurs and formal counseling is terminated (Hovell, Zakarian, Wahlgren, & Matt, 2000b). Whether these doses of exposure can be tolerated by medically at-risk children without acute symptomatic changes (i.e., are clinically significant) is not yet known. It is not yet clear how many sessions or the exact ''intensity'' of counseling that is needed to reduce the exposure to clinically important levels for the majority of patients for whom services might be appropriate. To determine dose effects, more expensive and much longer community trials would be necessary. For medically at-risk children for whom the greatest risks may be presumed, such studies might show the value of counseling over longer periods than is normally tested. Such studies are also needed to show the degree to which parents and children can tolerate the burden involved in long-term counseling programs or whether there is a threshold effect beyond

which no reduction in SHS exposure is likely. If such a threshold dose exists, it is likely influenced by the severity of the child's disease, concurrent exposure to other triggers, critical periods of exposure, and individual differences (Hovell et al., 2002a). Results to date do not provide guidance with regard to these issues. In the absence of an identified safe level of exposure for acute outcomes, it should be assumed that any level of cumulative SHS exposure is potentially toxic to children with medical problems.

Studies that provide feedback, such as inherent in repeated questionnaires, nicotine dosimeters in the home, or biochemical markers of SHS exposure (e.g., urinary cotinine), may inadvertently reduce SHS exposure in response to the measurement reactivity process (Hovell et al., 1994). Hovell et al. (1994) showed this effect with families of children with asthma assigned at random to minimal measures, full measures, and full measures plus counseling. The two measurement conditions resulted in >75% of the reduction in SHS exposure as that achieved by the full measures plus counseling. This is consistent with the literature showing that measures can be reactive and sometimes convey almost as large an effect on behavior as might be achieved by formal counseling or other interventions. It also highlights the potential use of self-monitoring as an intervention component in SHS clinical trials.

Two recent controlled clinical trials examined the efficacy of a feedback-based, parent-targeted intervention for families of children with asthma. Convincing reductions in SHS exposure were not reported. In one of these trials, parents were provided with verbal and written feedback about their child's baseline urinary cotinine levels, two telephone calls encouraging a ban on smoking at home, and information booklets about SHS-related health risks, compared to a usual care advice condition. No significant differences were found in the child's urinary cotinine levels, parental cessation rates, or restrictions in home smoking at 6 months between usual care controls or families assigned to the intervention group (Wakefield et al., 2002). In a similar study that included face-to-face counseling, Wilson and colleagues (Wilson et al., 2001) reported a 46% greater reduction in urine cotinine among children in the intervention group at a 12-month follow-up. The intervention–control differences were not statistically significant, which the authors attributed to decreased statistical precision secondary to loss of families to active follow-up.

However, there were fewer acute asthma-related medical visits in the following year among children in the intervention group compared to controls. Based on these collective results, it appears that providing limited objective feedback about levels of SHS exposure is not convincingly effective in motivating parents to modify their cigarette use. However, more intensive interventions that include repeated feedback and support over a longer duration may be necessary to determine the therapeutic effects for healthy and ill children.

Cessation Studies

Some success has been achieved in studies targeting parental smoking cessation as a means to control the child's exposure to tobacco smoke. Cessation studies have typically targeted parents of children visiting their pediatricians in the clinic setting but have not been extensively evaluated in medically at-risk populations. In one randomized trial that targeted women whose children were seen at pediatric clinics serving low-income families, self-reported abstinence rates were reported to be twice as great in an intervention group compared to a control group at a 12-month follow-up (Curry et al., 2003). The intervention included a brief motivational message from the child's clinician during the clinic visit, a self-help guide to quitting smoking, an in-person motivational interview with a clinic nurse, and up to three outreach telephone counseling calls from the nurse or study team. Several other randomized controlled trials have demonstrated small but significant increases in cessation rates among parents counseled about their smoking behaviors in an outpatient setting compared to controls (Severson, Andrews, Lichtenstein, Wall, & Akers, 1997; Wall, Severson, Andrews, Lichtenstein, & Zoref, 1995) or no effect (Groner, Ahijevych, Grossman, & Rich, 2000).

More recently, Winickoff, Buckley, Palfrey, Perrin, and Rigotti (2003a) demonstrated the feasibility and short-term efficacy of delivering tobacco dependence treatment for smoking parents at the time of their child's outpatient pediatric clinic visit. Participants were offered a three-session ''Stop Tobacco Outreach Program,'' which included motivational interviewing, written materials, nicotine replacement therapy (NRT), a referral to a free-state telephone quit line, and a fax referral to the parents' primary clinician. Of 100 participants, 56% reported making a quit attempt that lasted 24 hr, 18% reported 7-day tobacco abstinence, 34% used NRT, and 42% reported additional counseling from the state telephone quit line at a 2-month follow-up. Parents also reported smoking fewer cigarettes in their home and car over a 2-month period. Similar short-term efficacy was demonstrated at a 2-month follow-up for smoking parents

who received this same smoking cessation program during their child's hospitalization for a respiratory illness (Winickoff, Hillis, Palfrey, Perrin, & Rigotti, 2003b). Although these studies were limited in that they did not include a control group and relied on self-report measures, the high rates of parent acceptance of inpatient and outpatient counseling suggest that the child's medical care and contact with the health care system may provide appropriate opportunity to intervene with parents who smoke.

Special Considerations for Medically At-risk Populations

SHS reduction and cessation interventions, that have already been proven effective among parents of healthy children and those with asthma, may have applicability to other medically at-risk populations often seen in subspecialty clinics. Many of the studies conducted with children in general, to date, highlight some of the benefits of these approaches as well as some of the complications and obstacles that may be encountered in medically compromised populations. For example, current available SHS reduction or cessation approaches developed for the general population do not adequately address parent's informational needs about the effects of SHS on their child's specific health risks and vulnerabilities in the context of the child's disease and treatment. Nor do they capitalize on parental concerns about health protection (Mulhern et al., 1995) or heightened parental motivation to engage in behaviors to improve their child's health outcomes (Butz & Rosenstein, 1992; Winickoff, Hibberd, Case, Sinha, & Rigotti, 2001) that have been reported for some pediatric populations. There is evidence for decreased smoking or alterations in smoking behaviors (e.g., smoking outside) among parents of children with respiratory disease, including cystic fibrosis, following the child's diagnosis (Butz & Rosenstein, 1992). Likewise, data suggest that a child's hospitalization, regardless of the child's diagnosis, may advance parents' readiness to quit smoking (Winickoff et al., 2001). Parents of children with medical conditions, whose symptomotology is exacerbated by exposure to SHS and improved when exposure is reduced, may be further motivated to comply with smoking-based counseling if they are able to link changes in their child's symptomotology to exposure.

Therefore, revisions to the content and delivery, use of motivational strategies that capitalize on the parent's increased concerns regarding their child's health, and reliance on the supportive aspects of the treatment setting, may be necessary to enhance the impact of more traditional approaches when implemented with medically at-risk populations. Health care providers have the opportunity to repeat advice about eliminating exposure and cessation of smoking over several medical visits when parents are sensitized to their child's health. The assistance provided to parents by clinicians through direct clinical interventions may further enhance their motivation to protect their child from SHS.

Future Directions

More valid evaluations of intervention efficacy in future trials with at-risk pediatric populations will depend on improved measurement of SHS exposure and development of more sophisticated and reliable biological and environmental outcomes. Technological refinement of portable particle monitors worn by patients could provide advances in real-time and ongoing measurement of SHS exposure (Brauer, Hirtle, Hall, & Yip, 1999). In order to better study the relationship between disease and SHS exposure, it may be critical to obtain estimates of very subtle levels of exposure that can only be captured by highly sensitive measures.

Despite the moderate correlations between child cotinine levels and parent self-reports of exposure reported in the literature (Matt et al., 1999), not all trials have been able to confirm reported exposure using cotinine as an outcome (Greenberg et al., 1994; Wakefield et al., 2002). The use of cotinine as a biomarker has been controversial because it is limited by individual differences in uptake, distribution, metabolism, and excretion of nicotine (Matt et al., 1999). Additionally, few data are available at present to determine whether a child's treatment regimen and associated medications affect these processes and measurement of cotinine, if assessed during his/her active medical treatment. One study found that when compared to healthy children, children with asthma had significantly higher cotinine concentrations in the hair, despite lower parent-reported levels of exposure and urine cotinine (Knight, Eliopoulos, Klein, Greenwald, & Koren, 1998). This finding raises the possibility that there may be physiological differences in how children with asthma systemically respond to nicotine exposure. These potential pharmacokinetic differences, which may also exist for other diagnostic groups, deserve further consideration.

Another possibility might explain paradoxical cotinine results in SHS reduction studies. Matt et al. (2004) demonstrated low but non-zero levels of cotinine in children from homes of families who did not smoke.

However, families who smoked only outside the home were orders of magnitude higher in their child's cotinine level and nicotine contamination in the home; levels of SHS exposure and contamination were even higher for families who smoked inside the home. These findings suggest that nicotine may linger in the home. This provides the possibility that children could be exposed to SHS from off-gassing even when there is no ''active smoking'' in the home. It might also explain why some studies have found sharp reduction in parents' smoking around their children, but no effect on cotinine (or even an increase in cotinine levels). Two implications arise from these results. The first is that all homes and cars should be protected from SHS exposure, in order to better protect children. The contribution of exposure to off gassing to symptom exacerbation or later medical risk among medically compromised children has not yet been investigated. The second is that more research is needed to determine the degree to which home contamination might confound clinical results.

More recently, levels of tobacco-specific carcinogen metabolites have been examined in the urine of newborns and children exposed to SHS because of the suspected relationship between carcinogen uptake from SHS in childhood and the occurrence of lung cancer later in life. Levels of this carcinogen biomarker [total NNAL: 4-(Methylnitrosamino)-1-(3-Pyridyl)-1-butanol] have been found to be significantly correlated with cotinine levels in infants exposed to SHS (Hecht et al., 2006). To date, this biological measure has not been used as an outcome in pediatric SHS intervention studies. However, quantification of carcinogen uptake from SHS may be relevant to medically at-risk children, particularly those with cancer who received chemotherapy and/or radiation therapy, and are persistently exposed to SHS.

In addition to measuring observed changes in biomarkers, concurrent changes in clinical manifestations of the child's disease as well as acute and long-term health outcomes should be examined to better demonstrate therapeutic intervention effects. The dose–response relationship between reductions in exposure and improvements in disease outcomes is largely unknown. Modest reductions in exposure may have clinically significant health benefits for children such that failure to measure health outcomes as well as exposure may lead to underestimating the value of the intervention (Wilson et al., 2001). Longitudinal studies will be necessary to examine the long-term and cumulative effects of exposure reduction on children's health as it may take several years of reduced exposure to reverse the biological effects of years of high levels of repeated exposure (Wahlgren et al., 1997).

As most studies have focused exclusively on either SHS reduction or cessation, future trials should consider examining the combination of reduction strategies (i.e., smoking outdoors) with cessation counseling on both exposure reduction and cessation outcomes. One recent study showed higher cessation rates with use of any pharmaceutical aids (nicotine replacement therapy, bupropion or both) in homes with smoking bans (Gilpin, Messer, & Pierce, 2006). For parents who are ready to quit, cessation counseling, enrollment in a telephone quitline, and provision of pharmacotherapy may be acceptable (Winickoff et al., 2005, 2006); for those parents not yet ready to commit to smoking cessation, parental SHS reduction strategies may benefit their children. Rather than viewing modification of parent smoking behaviors and associated SHS reduction as an interim measure with cessation as the ultimate outcome, a combination of outcomes may be necessary. Total cessation by the target parent participating in the intervention may not guarantee total protection of the child's health, especially if there are multiple smokers in the home or parents allow others to smoke around their child. Therefore, behavioral interventions should target improvements on both children's health (via exposure reduction) and parent health (via smoking cessation) for most effective tobacco control (McQuaid, Walders, & Borrelli, 2003). Although parents have been the typical recipients of SHS interventions, educating older children about the effects of exposure on their disease and encouraging them to avoid exposure from adult and peer smokers is another area worth exploring.

Although learning theory, which emphasizes reinforcement and modeling, has provided an effective framework for understanding and changing SHS exposure practices (Bourland, 1999; Gehrman & Hovell, 2003), improved tobacco control efforts require expansion of this model beyond the individual level to emphasize the interaction of physiological, environmental, and cultural contingencies. A proposed ''behavioral ecological model'' (Hovell, Wahlgren, & Gehrman, 2002b) considers the density of reinforcement from the social networks of smokers and culture-wide social contingencies that may motivate parents to stop smoking around their child. Within the medical setting, clinicians are often the primary source of reinforcement for parent behaviors during a child's medical treatment and for that reason should incorporate SHS counseling into their clinical practices. Pediatricians, however, have not been confident

about their ability to modify the behaviors of parents who expose their children to SHS (Burnett & Young, 1999).

While clinicians may be able to initially evoke a reduction in SHS exposure, ongoing sources of reinforcement will help to sustain SHS reduction practices when parents are outside of the medical setting. A behavioral ecological approach requires the simultaneous implementation of many levels of intervention if substantial and cumulative reduction in child SHS exposure is to be achieved. Clinical programs, physician counseling, and behavioral counseling procedures for SHS reduction might yield significantly greater change if they were combined with multilevel, community-wide interventions and policies, media programs, and legislative action. Clinicians should be made aware of the potential of the cumulative effects of repeated interventions across settings and the population outcomes that are not readily apparent in the medical setting (Hovell, Wahlgren, & Russos, 1997). While behavioral research can provide parents with a repertoire of skills to reduce SHS exposure around their child, it must also begin to inform community-wide policies that will support these individual efforts for complete and effective tobacco control (Hovell & Daniel, 2005). In short, maintenance of child protection from SHS exposure and maintenance of smoking cessation will require a change in culture that involves continuous support for tobacco control.

Among future studies, some should be designed to contrast counseling programs in states with aggressive tobacco control legislation and taxation with states in which similar interventions are tested but for which much weaker tobacco control defines the context. Such cross-state studies will offer more complete understanding of the possible moderating effects of policies for clinical interventions. Ultimately, whole communities may be organized to cumulatively and synergistically provide tobacco control efforts that will reduce SHS exposure to children, especially those medically at-risk, as well as smoking rates population-wide. Until then, more focused research concerning the protection of the most vulnerable populations, medically at-risk children, should be supported to best understand how to protect them from SHS exposure.

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