Measuring Secondhand Smoke Exposure in Children: An Ecological Measurement Approach

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Objective *Behavioral, environmental, and biological measures of secondhand smoke (SHS) exposure are reviewed with special consideration of medically at-risk children*. **Methods** An ecological measurement framework is introduced to examine SHS exposure of children in the context of their physical and social environments. **Results** The proposed approach emphasizes the need to measure (a) who uses tobacco, (b) where and when exposure takes place, (c) what media are contaminated, (d) how exposure takes place, (e) how much a child was exposed, and (f) factors that contribute to why tobacco is used in a child's environment. **Conclusions** Existing research suggests that medically at-risk children are among the most vulnerable populations for the harmful effects of SHS exposure. Yet, little is currently known about how SHS exposure affects these populations. The proposed approach provides a framework for the comprehensive assessment of SHS exposure to study its health effects and to design effective interventions.

Key words children; contamination; exposure; measurement; secondhand smoke.

Secondhand smoke (SHS) is a complex and dynamic mixture of microscopic particles and gases, comprising more than 4,000 chemical substances (State of California Air Resource Board, 2006). Many of the individual components are known irritants, toxicants, mutagens, teratogens, and carcinogens in humans. Infants and children are known to be particularly vulnerable to the adverse health effects of exposure to SHS (U.S. Surgeon General, 2006). Based on a comprehensive review of the scientific evidence, the 2006 Surgeon General Report on the health consequences of involuntary exposure to tobacco smoke concluded: ''1. Secondhand smoke causes premature death and disease in children and in adults who do not smoke. 2. Children exposed to secondhand smoke are at an increased risk for sudden infant death syndrome (SIDS), acute respiratory infections, ear problems, and more severe asthma. Smoking by parents causes respiratory symptoms and slows lung growth in their children.'' (p. 11)

Although there has been a notable decline over the past 15 years, SHS exposure among children continues to be common. Data from the 1999–2000 National Health and Nutrition Survey (NHANES) indicate that 24.9% of children aged 3–11 years and 19.9% of adolescents and young adults aged 12–19 years lived in a household with at least one smoker (U.S. Surgeon General, 2006). Biomarker measurements of SHS exposure indicate an even larger population of exposed children. Based on serum cotinine measures, 59.6% (3–11 years) and 55.6% (12–19 years) may have been exposed to SHS in the year 2000. According to data collected between 2000 and 2007 as part of the Global Youth Tobacco Survey (Centers for Disease Control and Prevention, 2007), between 71.5% (Europe) and 22.5% (Africa) of adolescent never smokers were exposed to SHS over a 7-day period.

Due to the large number and complexity of toxins in SHS, numerous biologic mechanisms are suspected through which SHS causes injury and disease and why children in general and health-compromised children in particular are especially vulnerable. These include impairment of fetal airway development, the induction of

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bronchial hyper-reactivity, changes in the neural control and the balance of immune cells in airways, airway inflammation, inhibition of antibody responses contributing to impaired immune responses, inhibition of mucociliary clearance, disruption of respiratory epithelium, oxidative stress, inadequate cardio-respiratory compensatory motor responses, and sleep apnea attributable to developmental abnormalities in the brainstem and autonomic nervous system. Through these and other mechanisms, SHS exposure in utero and in early childhood can lead to morphologic and metabolic alterations that slow fetal growth, suppress fetal respiratory rate, reduce airway size, cause changes in lung properties, and interfere with the normal development of the lung. In addition to the damage inflicted on the developing lung, SHS can have a lasting impact on immune responsiveness, suppressing the ability to respond to a wide range of environmental challenges, and has been causally associated with some adult cancers such as premenopausal breast cancer (State of California Air Resource Board, 2006). SHS may also increase some immune responses, augmenting the potential for allergic sensitization, and may promote the development of an allergic phenotype (U.S. Surgeon General, 2006).

The increased vulnerability of health-compromised children is also due to important behavioral, social, and environmental factors. Infants in general have limited mobility and spend most time indoors at home and in close physical proximity to their parents (Klepeis et al., 2001; Wiley et al., 1991). This is especially the case in medically compromised children whose physical capabilities may be restricted, and who are cared for by family members at home, or undergo intrusive treatment regimens. Therefore, children are likely to spend considerable time in environments that are contaminated with SHS if parents or other family members smoke.

Young children, in general, spend more time on or near floors crawling or playing, putting them in closer proximity to dust contaminated with SHS constituents and surfaces on which SHS particulate matter may have accumulated. Children also have a tendency to insert in their mouths and swallow nonfood items, increasing the likelihood of ingesting contaminated objects. Because young children have higher breathing rates than adults, they breathe higher dosages of toxins than adults. Finally, children may be at a higher risk of exposure to SHS because public health policies aimed at protecting nonsmokers focus on locations of exposure that are more relevant for adults than children (e.g., workplace, restaurants, and airplanes). In contrast, the primary source of exposure for children are the parents, and most child exposure takes place in private settings such as homes and cars (Emmons, Hammond, & Abrams, 1994; Klepeis et al., 2001; Matt et al., 2000; U.S. Surgeon General, 2006), where smoking restrictions are voluntary and can be difficult to enforce.

With the exception of asthma, little research is currently available on SHS exposure among medically compromised children. Drawing on asthma as a case example, it is well known that SHS exposure can exacerbate symptoms in children (Cook & Strachan, 1997; Martinez-Donate et al., 2003; Strachan & Cook, 1998). Yet, even for asthma, we know very little about how SHS exposure might complicate medications, diagnostic outcomes, or otherwise confound the treatment of asthma. Similarly, we know very little about how treatment for asthma may confound studies of interventions designed to reduce SHS exposure. It is theoretically plausible that parents of asthmatic children might be more motivated to reduce SHS exposure if assisted by clinicians and some form of clinical intervention. Although studies on SHS exposure reduction with asthmatic children (including our own) have not addressed such motivation directly (i.e., by contrasting intervention effects for children not diagnosed with asthma), we have found remarkably long maintenance of effects after completion of the counseling intervention (Hovell et al., 2002a, Hovell, Wahlgren, & Gehrman, 2002b; Wahlgren, Hovell, Meltzer, Hofstetter, & Zakarian, 1997). We have speculated that the maintenance of reduced exposure after completion of the counseling intervention might be due to the natural ''biofeedback'' available to parents when they slip and expose their child to SHS. Under these conditions, the association between exacerbated symptoms and SHS exposure may be more easily discriminated as might the recovery of the child if immediately protected from SHS. This might ''teach'' parents to comply with procedures initially supported by counseling.

Unfortunately, there is little information about the complications involved in SHS exposure for children with other forms of medical compromise. O'Rourke, Kalish, McDaniel, and Lyons (2006) evaluated the association between SHS exposure and pulmonary function in 54 children aged 5–15 years both prior to and following anesthesia and surgery. They found significantly lower preoperative peak expiratory flow rates in the SHS-exposed children compared with nonexposed children, although the subsequent recovery from anesthesia for the two groups was similar. Among children aged 2–18 years

with sickle cell disease, exposure to SHS was found to be significantly associated with a higher risk of sickle cell crises than in nonexposed children after adjusting for several important covariates (West et al., 2003). Although children in general are known to be at greater risk of SHS exposure at home than are adults, only limited information is available on the routine exposure to SHS among hospitalized children. In a cross-sectional survey of SHS exposure among 848 children in the general pediatric wards of four Hong Kong hospitals reported in 1999, Chan et al. (1999) found that 86% of the children in that study lived with 1–3 smokers who smoked at home, and 61% lived with smokers who smoked near them at home nearly every day. In each of these studies, exposure was based on surveys and interviews of parents and other caregivers; additional studies of SHS exposure in medically compromised children that are based on the use of objective biomarkers would be beneficial.

Children with cystic fibrosis (CF), a chronic and life limiting pulmonary and digestive disease, might suffer considerably from SHS. They also are treated with pulmonary medications similar that of asthmatic children to aid their ability to breathe, and they routinely take antibiotics for infections and enzymes to aid in digestion. To our knowledge, there is no research that informs the degree to which treatment or diagnostic procedures are complicated by SHS exposure or the degree to which the prognosis for children with CF might be enhanced with reduced exposure. Essentially, the same is true for children undergoing acute treatment for cancer (Tyc et al., in press). This list goes on for diabetes, severe injury, and other forms of morbidity. As noted by Tyc (2005), we know that cotinine assays are feasible for children undergoing chemotherapy, but we also know that the urine samples do not resemble normal urine. Results from the current trial for these children may inform more than all other studies to date how chemotherapy might alter counseling effects or measurement of SHS.

In this context, this article provides a summary of the state of the art measures for SHS exposure but does so for children in general as this is the population that most of the research to date reflects. Future studies will be needed to determine the possible interactions and complications involved in measures of SHS exposure for medically compromised children. This is especially important from an ecological perspective, where the pre-existing disease and medical care represent context variables of potentially profound influence on both SHS exposure control and assays designed to measure SHS exposure.

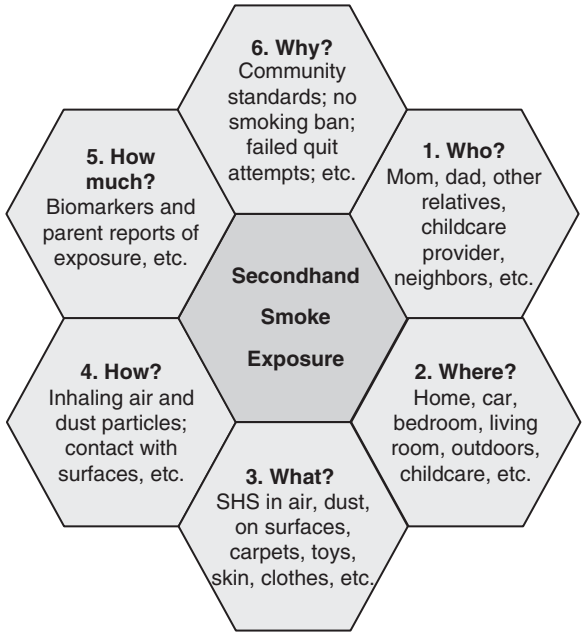

Figure 1. An ecological measurement approach to SHS exposure in children.

The proposed measurement model addresses SHS exposure of children in the physical and social environments in which children live. Because of the large number of suspected biological mechanisms through which SHS can cause harm and our interest in health compromised children, the proposed model pays attention to multiple exposure pathways, low dosage and occasional exposure, exposure during active smoking and exposure to lingering residual contamination, and exposure opportunities in different locations and from multiple sources throughout the social and physical environment of a child. Thus, the proposed ecological measurement model goes beyond examining whether and how much SHS exposure took place. We argue that it is also important to assess where, who, how, and why SHS exposure takes place so that appropriate actions can be taken to intervene in the physical and social environments of children to reduce, remove, avoid, and eventually prevent exposure to SHS. Such a model is particularly suited for future clinical research on health compromised children for whom even relatively low levels of SHS pollutants may exacerbate an illness and interfere with treatment and recovery.

Figure 1 provides an overview of the proposed model. Asking questions about *who* uses tobacco in the immediate family and extended social network, we can identify the potential sources of SHS. By asking questions about *where and when* exposure takes place, we can identify the locations that are most contaminated with

SHS and on what days, occasions, and time of day SHS levels are likely to peak. Collecting environmental samples (e.g., air, dust, and surfaces) to measure SHS contamination allows us to determine exactly *what* media are contaminated in a child's environment. By asking questions about *how* exposure takes place, we seek to identify the pathways by which SHS pollutants enter a child's body. Collecting biological samples (e.g., urine, blood, saliva, and hair) allows us to quantify *how much* exposure took place. Finally, we need to ask questions about *why* a child is exposed to understand contingencies in the child's physical and social environment that maintain tobacco addiction in family members and the pollution of the child's environment with SHS.

Who Smokes in a Child's Social and Physical Environments?

A convenient starting point for measuring the exposure of children to SHS is the systematic assessment of the smoking behavior of persons in the social and physical environments of children. This includes persons with whom children interact and persons who use the same physical environments where children spend time. Both are important in their own right because each represents a potential source of exposure (Daisey, Mahanama, & Hodgson, 1998; Matt et al., 2004; Singer, Hodgson, Guevarra, Hawley, & Nazaroff, 2002).

The most direct exposure pathway is when a child inhales SHS while someone smokes. It is therefore no surprise that a smoking mother is the most important source of SHS exposure in young children (U.S. Surgeon General, 2006). The same is also likely to be true for older children and adolescents with medical conditions who are cared for by a smoking mother.

There is new evidence that SHS exposure can even occur in the absence of active smoking in the immediate proximity of a child (Matt et al., 2004; Nazaroff & Singer, 2004). Tobacco easily disperses from room to room in an apartment and can drift from a neighboring apartment or an outdoor smoking area into a home. This is why restricting smoking to a particular room in a home does not prevent SHS from spreading through the entire home and does not protect children from SHS exposure (Klepeis & Nazaroff, 2006a,b; Singer et al., 2002).

SHS gases and particles also sorb into surfaces and become trapped on the clothes and skin of smokers. From there, pollutants can be re-emitted later as ''off gasses,'' leading to SHS exposure long after cigarettes have been smoked. Children may be exposed to components of SHS produced by persons with whom they have never interacted face-to-face, but who smoked in an environment shared with children. This can occur when smoking takes place at a time when children are absent, and SHS pollutants sorb and deposit on surfaces and in dust that remain in the environment long after smoking has ceased. Children may then be exposed to this lingering SHS (also known as thirdhand smoke), when they enter such a space and come in contact with the residual contamination (Aitken, Kenny, & Soutar, 2001; Szabo, 2006).

The most common method of assessing smoking behavior in the social context of a child is through personal interviews, questionnaires, and behavioral diaries (Hovell, Zakarian, Wahlgren, Matt, & Emmons, 2000c; Pechacek, Fox, Murray, & Luepker, 1984). These instruments often rely on a parent's or other caregiver's report when the child is young. The administration of questionnaires is noninvasive and relatively inexpensive, may easily be repeated over time, and provides the only effective means of collecting retrospective data, including those addressing smoking history. Such measures are also the best means of assessing qualitative social and physical contexts and possible sources of confounding events that might compromise biological markers of exposure. For instance, questionnaire measures are the most efficient means of learning about rules banning smoking in the home and any exceptions, for instance, for the child's grandfather as a result of complex interpersonal relationships with the child's parents.

Self-reports and parental questionnaires are necessarily subjective, in that they reflect potential biases of a specific respondent. Interview measures can be very demanding because they require excellent memory and strong verbal skills that may exceed those of the respondent. They can be too simplistic providing only very indirect or incomplete proxy measures of the construct of interest. This can contribute to recall errors, or systematic under or overestimation of smoking behavior (Matt et al., 1999). Interview measures may also have reactive measurement properties in that they may influence what they are supposed to measure (Webb, Campbell, Schwartz, Sechrest, & Grove, 1981). For instance, detailed questions about who, where, when, and how many cigarettes are smoked by all members of a household my help a parent understand the scope of the problem and suggest ways to better protect a child from exposure.

Regardless of which approach is taken to measure tobacco use, the goal in the context of the proposed ecological measurement approach is to describe who are

the persons in a child's environment who smoke, and to determine how much, how often, and where they smoke over a representative time reference period. The usefulness of interviews depends on the reliability and validity and the extent to which the smoking behavior is causally connected to actual contamination of microenvironments and biological exposure of a child. There are numerous studies of the reliability and validity of different measurement methods for assessing smoking behavior, demonstrating that well-designed personal interviews, questionnaires, and behavioral diaries can have excellent reliability and validity (Dolcini, Adler, Lee, & Bauman, 2003; Patrick et al., 1994; Studts et al., 2006). For instance, Matt et al. (2000) report a test–retest reliability for the mother's reported indoor smoking rate of .91, and criterion-related validity coefficient of .62 (infant urine cotinine) and .75 (air nicotine level). Such desirable measurement properties, however, should not be assumed but systematically investigated through appropriate psychometric analyses whenever smoking behavior and SHS exposure are assessed.

Where and When Does Smoking Take Place?

Because smoking contaminates the physical environment of a child with pollutants (State of California Air Resource Board, 2006), it is important to assess whether a child spends time in locations and comes in contact with media (e.g., air, dust, and surfaces) that are contaminated with SHS. To establish the whereabouts of a child during a representative period of time, a time-activity diary can be very useful (Hovell et al., 2000c). Time-activity diaries establish the indoor and outdoor spaces in which the child was present (including living room, patio, car, playground, and bedroom), how much time was spent there, who the child interacted with, and the types of activities in which the child was engaged (e.g., playing, sleeping, and sitting). Based on such a schedule, one can then determine if smoking took place in these locations in the presence or in the absence of the child and how much time the child may have spent in a contaminated environment.

Because smoking behavior varies over time, it is important to recognize that its assessment on any specific day can only provide a snapshot that may not be representative of typical smoking behavior in the environment of a child. Therefore, it is important to choose a time reference period that is sufficiently large to capture important variability in smoking behavior. This includes workdays and nonwork days, birthdays, sick days, school days, days with doctor's visits, vacation, and seasonal variation.

What is Contaminated with SHS in the Physical Environment of a Child?

SHS consists of a mixture of the smoke given off by the burning end of a cigarette, pipe, or cigar (i.e., sidestream smoke), and smoke exhaled from the lungs of smokers (i.e., the mainstream smoke). Sidestream smoke makes up about 85% of SHS and burns at lower temperatures than mainstream smoke. This causes sidestream smoke to contain higher concentrations of toxic gases and smaller particles than mainstream smoke (California Environmental Protection Agency, 1997; Jaakkola & Jaakkola, 1997).

SHS pollutes the ambient air with particulate matter (PM), volatile organic gases, and gas-phase inorganic compounds. PM of SHS is produced at about 7–18 mg per cigarette (Daisey, 1999; Klepeis, Apte, Gundel, Sextro, & Nazaroff, 2003; Leaderer & Hammond, 1991) and consists of the condensed (as opposed to the gaseous) phase of a pollutant. Emission and ventilation rates have the largest influence on indoor air concentrations of PM. The mass median aerodynamic diameter of SHS particles is $\sim$0.2 μm with a wide range from 0.01 to 1.0 μm. The size of particles determines whether and where they deposit in the lung. PM of SHS contributes significantly to respirable suspended particulate matter (RSP), defined as particles $<$2.5 μm mass median aerodynamic diameter (Willers, Schutz, Attewell, & Skerfving, 1988; Willers et al., 1992) that can deposit deep in the lung (i.e., alveoli). Real-time RSP concentrations are relatively easy to measure with commercially available monitors (e.g., TSI SidePak AM510 Personal Aerosol Monitor, TSI, Inc., St Paul, Minnesota, USA) and do not require subsequent biological and chemical laboratory analyses.

Although RSP are not specific to SHS, they are strongly correlated with other SHS pollutants (California Environmental Protection Agency, 1997; Repace, Al-Delaimy, & Bernert, 2006a). Because of their harmful properties, the fraction of fine-particulates (PM2.5) is a regulated ambient air pollutant and forms the basis of the widely used outdoor Air Quality Index (AQI). RSP concentration increases with the number of cigarettes being smoked, and it is common that RSP concentrations exceed AQI levels deemed ''very unhealthy'' ($>$150 μg/m^3) and ''hazardous'' ($>$250 μg/m^3) in indoor environments where smoking occurs regularly (Repace, Hyde, & Brugge, 2006b). It should be noted that the RSP concentrations in the ''unhealthy'' and ''hazardous'' ranges are more than 34 and 56 times higher than the irritation threshold of 4.4 μg/m^3 reported by Junker et al. (2001). This suggests that these standards may allow

more exposure than is safe, especially for children or medically compromised children.

In addition to PM, Daisey (1999) discussed the importance of volatile organic compounds and gas-phase inorganic compounds (e.g., CO). The indoor air concentration of highly volatile organic compounds (e.g., formaldehyde) is largely determined by emission rates, indoor volumes and ventilation, with sorption and re-emission playing only a minor role. For semivolatile organic compounds (SVOC; e.g., benzene, nicotine, and pyrene), however, sorption on and desorption from surfaces can have a major impact on air concentration. Indoor surfaces act as reservoirs or sinks for this class of volatile organic compounds from which they are re-emitted over time. In indoor environments in which smoking occurs regularly over an extended period of time, the sorbed mass of these compounds can become large relative to the mass emitted by a single cigarette (Daisey, 1999; Nazaroff & Singer, 2004; Singer et al., 2002). Consequently, re-emission of these compounds from indoor surfaces may become significant relative to direct emission. In a study of the effect of room furnishings on the emission rate of toxins from cigarettes, sorption of SVOCs into room furnishings was found to be significant for many measured SHS compounds such as cresols, naphthalene, nicotine, and 3-ethenylpyridine (Singer et al., 2002). Consequently, these surfaces may represent a significant reservoir for the re-emission of SHS, including some of the more toxic components of tobacco smoke. Unfortunately, the measurement of specific SHS pollutants in the air, on surfaces, or in dust can be costly, requiring specialized equipment and expertise in the collection and chemical analyses of environmental samples (Hammond & Leaderer, 1987; Hammond, Leaderer, Roche, & Schenker, 1987; Matt et al., 2004; Singer et al., 2002).

In summary, SHS is not just diluted sidestream and mainstream smoke that is produced near a smoker while cigarettes are being smoked. SHS can travel through the air from room to room or from an outdoor smoking area through a window into an adjacent home. SHS can also travel on the clothes and skin of smokers or in the contaminated interior of a car. SHS contamination can persist over time because pollutants deposit on surfaces, mix with dust, and are re-emitted into the air from the surfaces to which they adsorbed.

Environmental Markers of SHS Contamination

Several constituents of SHS have been used as atmospheric markers of contamination, including such quantitatively significant compounds as carbon monoxide (CO), RSPs, solanesol, 3-ethenylpyridine, and nicotine. Although nicotine is initially associated with the particulate phase of tobacco smoke, it rapidly transfers to the gas phase and is mainly a vapor-phase component in SHS (Daisey, 1999). CO and RSP have limitations in assessing passive tobacco exposures because they lack specificity, whereas 3-ethenylpyridine, solanesol, and nicotine are all regarded as specific markers of SHS. Atmospheric markers of SHS have been monitored with both active methods using sampling pumps, and more commonly with simple, passive area monitors and individual badges for measuring nicotine (Hammond & Leaderer, 1987; Hammond et al., 1987). Badges may have some limitations in studies of younger children, but several studies of children's exposure have used nicotine area monitors effectively in specific locations in the home (Berman et al., 2003; Gehring et al., 2006; Hovell et al., 1994, 2000a).

Environmental monitoring of compounds specific for SHS such as nicotine area monitors provides a direct index of the *potential* exposure of the child to vapor-phase nicotine, and by extension, to SHS. Such analyses provide an objective, integrated index of nicotine concentration in the room over time, a direct indication of the markers' mean air concentration that is not subject to the metabolic variability that may influence biomarker concentrations, and are usually relatively simple to conduct. However, area exposure estimates of particular microenvironments may require extensive coordination with the child's time-activity patterns to develop a final exposure estimate (Jaakkola & Jaakkola, 1997), and passive-diffusion devices which are the type most commonly used have low diffusive flow rates and thus relatively low sensitivity, requiring integration of exposures monitored over several hours or days. Nevertheless, measuring specific atmospheric markers, especially the measurement of air nicotine and RSP, are crucial to the measurement of SHS contamination (Repace, 2007).

Studies of children's exposure to SHS estimated from vapor-phase nicotine collected in passive diffusion devices over a period of several days in a defined area or areas of the home have consistently shown significantly higher air nicotine concentrations in homes where smoking is permitted in comparison with nonsmoking homes (Berman et al., 2003; Gehring et al., 2006; Matt et al., 2004). Matt et al. (2004) showed that dust and surfaces that had been contaminated with SHS may also be sources of nicotine and presumably other SHS components in homes where smoking occurs. Air nicotine concentrations tend to correlate with biomarker levels, even though both measures may demonstrate

considerable variability in response to quantitative exposure metrics such as the number of cigarettes smoked per day in the home, or reported hours of exposure (Matt et al., 2007). For instance, Matt et al. (2000) observed a correlation of .74 between average air nicotine levels measured over the course of 1 week in the home of a smoking mother and the cotinine concentration in her child's urine (i.e., an established biomarker of SHS exposure).

Area measurements of atmospheric markers provide a general index of contamination from SHS within a defined microenvironment. Personal monitoring methods can extend this to a contamination index integrated over time and several microenvironments as experienced by the subject, although personal monitoring of air nicotine has seldom been used in studies of children's exposure to SHS. In either case, however, the measurement is only of potential exposure. To confirm that the compound was actually absorbed by an individual, and to obtain an index of the dose, the measurement of biomarkers is required.

In addition to atmospheric measures of SHS contamination, there is evidence that SHS also contaminates surfaces and dust in homes and cars as well as the skin and clothes of smokers (Hein, Suadicani, Skov, & Gyntelberg, 1991; Matt, 2007; Matt et al., 2004). This creates the opportunity that SHS exposure, especially in children, may also take place through hand-to-mouth transfer and ingestion. There is some initial evidence that dust and surface contamination significantly contributes to overall exposure in infants of smoking mothers (Matt et al., 2004). The re-emission of sorbed SHS pollutants into the air can sometimes be detected as a lingering stale tobacco odor. Because of the causal association between tobacco odor and SHS exposure (U.S. Surgeon General, 2006), further research on the nature and potential health effects of lingering SHS is warranted, especially in populations that may be vulnerable to low levels of SHS contamination.

Similar to smoking behavior, SHS contamination is likely to be highly variable over time and locations. As discussed earlier, common atmospheric measures of SHS contamination provide aggregated indices over time and microenvironments. While it is desirable to characterize average contamination levels, such indices necessarily ignore that contamination levels at the time smoking occurs may be many magnitudes higher than the integrated average for periods of time. Similar to real-time measurement of PM concentrations, it would be desirable to develop measures for the real-time measurement of nicotine, solanesol, and 3-ethenyl pyridine. Moreover, with the exception of air contamination relatively little is currently known about the spatial variability of SHS contamination in a home.

Interview Measures of Environmental Contamination

If personal interviews or questionnaires ask about smoking behavior, it is relatively easy to yield a proxy measure of the contamination of specific environments with SHS. This can be accomplished by decomposing overall questions about smoking into smoking in different locations (e.g., living room, bedroom, car, and patio). For instance, Matt et al. (2000) asked parents to estimate the number of cigarettes smoked at home and in the family car. These questions can be further decomposed into whether cigarettes are smoked in the living room, bedroom, bathroom, etc. Similarly, interview questions could ask about smoking in the home and cars of friends where the child may have spent time, or at the home of a childcare provider. Matt et al. (2000) demonstrated that a measure of reported cigarette smoking at home that was composed of separate questions for different smokers and workday and nonworkday smoking can show moderately strong agreement with air nicotine levels ($r = .35–.69$) and infant urine cotinine ($r = .52–.59$).

Similar to interview measures of smoking behavior, environmental, and interview measures of SHS contamination are likely to have reactive measurement properties. That is, wearing a personal air monitor or placing a stationary monitor in a home draws attention of smokers and nonsmoker to the tobacco use and SHS exposure. This may sensitize nonsmokers to the smoking behavior of household members and its potential effect on their children, potentially causing the nonsmoker to ask a household member not to smoke or to open a window or to move the child to a different room. These monitoring devices may also sensitize a smoker to smoke in a room without a monitor or to smoke less. To reduce the potential for reactivity biases, Hovell et al. (1994, 2000a, 2002a) have placed inactive monitors that cannot be distinguished from active monitors in all rooms of a home.

How are Children Exposed to SHS Contaminants?

Exposure occurs when a child comes in contact with contaminated media. The dose of exposure refers to the amount of a contaminant that crosses a boundary of the

body (Jaakkola & Jaakkola, 1997). The two best-known sources of postnatal SHS exposure in children are via inhalation while in the presence of a smoker and from ingestion of breast milk (California Environmental Protection Agency, 1997). This may create the impression that bottle-feeding, smoking in the absence of a child, or smoking outdoors or near an open window could protect children from SHS exposure. As our review of the chemical and physical properties of SHS pollutants has shown, smoking not only contaminates the air in the immediate vicinity of a smoker, but can lead to long-term and wide-spread contamination of indoor air, dust, and surfaces, including the clothes and skin of the mother.

Inhaling SHS Contaminated Air

SHS may be inhaled when the smoking person is present or in another room, and long after smoking has taken place. The relationship between air nicotine levels at home and SHS exposure in children is well established (Chilmonczyk et al., 1990; Greenberg et al., 1991; Henderson et al., 1989; Matt et al., 1999, 2000).

It has been well documented that establishing a designated smoking area in a home does not eliminate the risk of SHS exposure. Particularly in small homes and apartments, tobacco smoke easily spreads throughout the rooms (Repace, 2007; U.S. Surgeon General, 1986). Volatile components of SHS are sorbed on surfaces and re-emitted long after a cigarette is smoked. Part of the PM component of SHS eventually settles out and becomes part of household dust, collecting in carpets, on furniture, and toys. Even if rooms are well ventilated, carpets, walls, doors, etc. are reservoirs of SHS from which SHS is re-emitted weeks and months later. That is, a child may inhale, ingest, or have skin contact with SHS many days or weeks after a parent or visitor has smoked and even if she was not present in the room at that time.

Inhaling SHS Contaminated Dust

Vapor and particle phase components of SHS can contaminate household dust, presenting yet another source of exposure. Infants and children who remain in an indoor environment where smoking has occurred may inhale SHS contaminated dust. Since infants and young children typically spend more time indoors and are in closer proximity to and engage in greater activity in areas where dust often collects (e.g., carpets on the floor) than adults, they are at increased risk for SHS exposure through contaminated indoor dust.

The first study to examine nicotine in household dust was conducted by Hein et al. (1991) who found a strong positive correlation ($r = .65$) between smoking rate and nicotine concentration in the house dust of 34 smokers and 38 nonsmokers. These data demonstrated that nonsmokers may inhale tobacco components from respirable dust, even if smoking does not occur. The amount of nicotine inhaled during 1 hr was estimated for someone in a home with high nicotine concentration in the house dust to be 12 ng, a relatively small amount compared to that inhaled by an active smoker (i.e., 600–3000 ng/hr). However, because infants and health compromised older children may spend the entire day indoors inhaling contaminated respirable dust, and have a higher respiration rate (factor 3–8) and a lower body weight than adults (factor 10–20), this relatively low dosage of SHS exposure may accumulate over the course of weeks to levels equivalent to several hours of active adult smoking.

Hand-to-Mouth Transfer of SHS Contaminants

Indirect ingestion exposure is generally defined as mouth and tongue contacts with a contaminated object. Hand-to-mouth transfer is recognized as a potent pathway for lead exposure in children and adults (Mielke & Reagan, 1998). SHS emitted from a cigarette sorbs on surfaces such as skin, toys, clothes, bed frames, tables, and walls. SHS contaminated dust settles on carpets, dishes, bottles, toys, clothes, etc. During childhood, mouthing and sucking are a normal and important part of development. Through mouthing, children explore their environment, obtain a sense of security and comfort, and seek pleasure. During this stage of development, children put their hands and any object that they come in contact with into their mouths. Therefore, young children exhibit a much higher frequency of mouthing behavior (i.e., mouth-to-hand, -body, -toy, -surface) and ingestion of nonfood items (i.e., pica behavior) than older children or adults (Cohen Hubal et al., 2000; Tulve, Suggs, McCurdy, Cohen Hubal, Moya, 2002). Tulve et al. (2002) found that children under 24 months exhibited the highest frequency of mouthing behavior with a median frequency of 73 events per hour. Children between 24 and 60 months exhibited a median frequency of 31 events per hour. That is, in addition to increased inhalation of contaminated dust, young children may be exposed to SHS through ingesting and touching contaminated objects and surfaces (e.g., toys, clothes, and pacifiers).

SHS components also contaminate a smoker's skin and clothes (Cieslak & Schmidt, 2004; Matt et al., 2004). If no precautions are taken (e.g., changing clothes, wearing gloves), young children may be exposed to tobacco components through sucking on a smoker's fingers or drinking from a bottle handled by a smoker.

Dermal Transfer of SHS Contaminants

Dermal transfer is recognized as an important pathway for exposure to pesticides (Lu & Fenske, 1999), semi-volatile chemical compounds (Krieger et al., 2000), polychlorinated biphenyls (PCBs), and polycyclic aromatic hydrocarbons (Nawrot et al., 2002; Wilson, Chuang, Lyu, Menton, & Morgan, 2003). The latter include several known human carcinogens that are also constituents of SHS.

While there is growing evidence of SHS contamination of household surface, dust, and the clothes and skin of smokers, we are not aware of any research on SHS exposure focusing on hand-to-mouth, ingestion, or dermal transfer in children. Because children are likely to be at higher risk of exposure to these sources and may have lower tolerance to SHS toxins, future research on these exposure pathways is warranted.

How Much Exposure is Taking Place?

Dose of exposure is a function of the concentration of a pollutant, the time course of exposure, the physiological state of the individual, and the pathway through which a pollutant enters the body (e.g., inhalation, ingestion, and transdermal). As we discussed earlier, the physiologic state, the development stage, and the medical condition of a child may alter the dose of exposure relative to that of adults who live in the same environment.

Biomarkers of SHS Exposure

For exposure to occur, the child and the pollutant have to be simultaneously present at a particular location and at a specific time. To confirm that SHS was actually absorbed by a child, and to obtain an index of the dose, the measurement of biomarkers is required.

Biomarkers of SHS exposure are SHS components or their metabolites measured in human tissues or physiologic fluids. Measurement of SHS-specific biomarkers can confirm exposure and may also contribute to an estimate of dose. A substantial number of compounds have been studied as potential biomarkers including carbon monoxide (in exhaled air or as carboxyhemoglobin), thiocyanate, nicotine, and its metabolites (e.g., cotinine), tobacco-specific nitrosamines, volatiles such as 2,5-dimethylfuran, several adducts of serum albumin or of hemoglobin, and other compounds (U.S. Surgeon General, 2006). Because of limitations in both sensitivity and selectivity for SHS exposures, CO and thiocyanate are less frequently used as exposure biomarkers now than in the past (U.S. Surgeon General, 2006). Cotinine, the primary proximate metabolite of nicotine, is currently regarded as the biomarker of choice because it combines excellent sensitivity and specificity for SHS exposure (Benowitz, 1996), although cotinine's relatively short half-life in the body of 16–18 hr limits the exposure interval to only the previous few days. This half-life is approximately the same in serum, urine, and saliva (U.S. Surgeon General, 2006).

In general, the preferred matrix for cotinine analysis is serum (Benowitz, 1996; Watts, Langone, Knight, & Lewtas, 1990). Cotinine in serum is stable for at least several years when samples are stored frozen, and it has been measured in a variety of ways as described subsequently. More recently, there has been interest in measuring both cotinine and its metabolite *trans*-3′-hydroxycotinine, since the hydroxycotinine/cotinine ratio may serve as an index of nicotine metabolizing activity in an individual (Dempsey et al., 2004). However, measurement of cotinine alone is sufficient for assessing exposures to SHS (Matt et al., 2006), especially for younger children. US national exposure estimates have consistently found children to have significantly higher serum cotinine concentrations than nonsmoker adults (U.S. Surgeon General, 2006). Although differences in pharmacokinetic parameters such as respiration rate and daily urine volumes may influence measurements such as urinary cotinine in very young children (Repace et al., 2006a), the relatively higher serum cotinine concentrations in children are believed to reflect their greater risk of daily exposure to SHS, particularly in the home.

In studies requiring repeated measurements and a noninvasive source for cotinine, either saliva or urine might be used. Saliva is an excellent matrix for cotinine exposure measurements (Etzel, 1990), and it has been used in a number of studies. Concentrations of cotinine in saliva are typically similar to but slightly higher than serum concentrations, and cotinine concentrations in the two matrices tend to correlate closely to each other (Bernert, McGuffey, Morrison, & Pirkle, 2000). Collection of saliva samples is relatively easy to accomplish except in infants, but volumes may be limited in some cases, and care must be taken to avoid contamination. Urine is another noninvasive matrix for cotinine measurements,

and it is probably the most commonly used matrix for SHS exposure assessments of interventions designed to reduce infants' and young children's exposure. A significant advantage of urine is that the concentration of cotinine and other nicotine metabolites is several-fold higher in this matrix than in serum or saliva, which can provide enhanced sensitivity for detection of low-level exposures to SHS. However, urine contains a complex mixture of nicotine metabolites including the glucuronides, and cotinine glucuronide may be the major form of cotinine present in some samples. Some methods hydrolyze the glucuronides before measuring the total cotinine concentration, whereas others measure only the free component. Unlike cotinine itself, cotinine glucuronide may not be stable in urine during storage at higher temperatures or following repeated freeze-thaw events (Hagan, Ramos, & Jacob, 1997). Free cotinine is the breakdown product, however, so differences resulting from loss of the glucuronide would not be expected when total cotinine measurements are made.

Another potential disadvantage of urine cotinine assays is the variability in urine dilution resulting from differences in hydration. Some investigators have attempted to adjust for this effect by using a correction for urinary creatinine concentrations (National Research Council, 1986; U.S. Surgeon General, 2006), most commonly by calculating a cotinine–creatinine ratio. However, this approach assumes similar glomerular filtration and tubular reabsorption characteristics of the two markers, which may not be valid, and it is sensitive to differences in creatinine concentrations in infants and young children. Thus, whether creatinine adjustments can significantly improve urinary cotinine measurements in children as an index of SHS exposure remains uncertain. Since concentration estimates may differ significantly according to the analytical source, especially in comparing urine with other matrices, a means of interconverting results from different studies would be helpful, and Repace et al. (2006a) recently provided a set of equations that can be used to interconvert cotinine results measured in serum, saliva, or urine.

Because of the short half-life of cotinine and the consequently limited exposure period that it can monitor, there is interest in alternative exposure markers of potentially greater duration, and hair nicotine or cotinine has been proposed for this purpose. The attraction of hair analyses is that the systemic deposition of exposure markers such as nicotine or cotinine into the growing hair shaft can provide an integrated record of exposure over time (U.S. Surgeon General, 2006). For example, assuming an average hair growth rate of approximately 1 cm/month, the typical analysis of a 3 cm segment of hairs proximal to the scalp could provide a mean exposure estimate extending over the prior 3 months. Unlike other matrices, hair assays have more commonly measured nicotine rather than cotinine since presumably both are stable in the hair shaft, and the former marker is in higher concentration in hair, although cotinine can also be measured in hair and it has been included in some studies. Several investigators have reported useful results with this approach in children, and the use of hair analysis for SHS exposure assessment was recently reviewed (Al-Delaimy, 2002).

Hair samples and questionnaire information on SHS exposure were collected by Nafstad et al. (1995) from 94 children aged 12–36 months. Compared to children with no reported exposure to SHS, hair nicotine concentrations were nearly four times higher in children exposed to the smoke from an average of 1–10 cigarettes per day in the home, and 12 times higher in those exposed to more than 10 cigarettes per day. In a study of 164 children including 78 with asthma, the asthmatic children had a 2-fold higher hair cotinine concentration despite being exposed to fewer cigarettes per day according to parental reports (Knight, Eliopoulos, Klein, Greenwald, & Koren, 1998). Al-Delaimy, Crane, and Woodward (2002) conducted a cross-sectional survey of 322 children, aged 3–27 months, admitted to hospitals in New Zealand for lower respiratory illnesses. Hair nicotine levels were correlated with the number of smokers in the home and with household cigarettes smoked per day, and hair nicotine was found to be correlated with questionnaire smoking variables ($r^2 = .55$). These results suggest that hair nicotine and/or cotinine measurements can provide a useful index of SHS exposure in children.

However, several questions remain concerning hair analyses, including the selection of representative hair samples for analysis and the influence of the melanin content of hair on nicotine deposition (U.S. Surgeon General, 2006). The main limitation concerns the uncertain source of nicotine in hair, because although systemic deposition certainly occurs, direct adsorption of nicotine from the environment onto the hair shaft also occurs. Investigators typically wash hair samples prior to analysis, but different approaches have been used and the effectiveness of the washing procedures remains uncertain. Either internal deposition or surface adsorption of nicotine would represent an exposure to SHS, but only the former would reflect an integrated dosage over time, whereas the latter would actually reflect a form of

personal atmospheric monitoring that might have occurred over a limited time period. Since cotinine is primarily a metabolite rather than an environmental contaminant, the analysis of hair cotinine might help circumvent the uncertainties in this assay, but cotinine concentrations in hair are much lower than nicotine, and studies based on the assay of cotinine have generally been less useful than nicotine (U.S. Surgeon General, 2006). It should be noted that although at much lower concentration than nicotine, cotinine has also been detected in SHS (Eatough et al., 1989). Hair analysis would seem to offer promise for SHS exposure assessments in children, but additional validation work is needed with this matrix.

Several methods are available for the analysis of cotinine as a biomarker of SHS exposure. Immunoassays are commonly used and can provide a rapid and relatively inexpensive analysis that is well suited to larger studies. Radioimmunoassay methods have good sensitivity and are still used in a few laboratories, but newer enzyme-linked immunosorbent assays based on monoclonal antibodies are now readily available commercially and are quite convenient. Potential limitations of these assays include the risk of cross-reactivity, and the limited sensitivity of typical ELISA for low-level exposures, although the lower sensitivity can be ameliorated somewhat by using urine with its inherently higher cotinine concentrations as the matrix. Recently, simple immunochromatographic devices in a ''dipstick'' format have become available for detecting cotinine in urine or saliva (Bernert, Harmon, Sosnoff, & McGuffey, 2005; Gariti et al., 2002). These devices provide only a semi-quantitative estimate of exposure, but they appear to be reasonably accurate, can provide immediate feedback, and they are quick and simple to use in a nonlaboratory environment. However, more information is needed concerning their validity in assessing exposures in children, especially in older children in whom occasional tobacco use may be a confounder.

A number of chromatographic methods for the analysis of cotinine are also available (U.S. Surgeon General, 2006), commonly using gas chromatography linked to either a nitrogen-specific detector or to a mass spectrometer, or liquid chromatography (LC) with either UV or mass spectrometry used for detection. Over the past few years, the use of LC with atmospheric-pressure ionization tandem mass spectrometry has become increasingly available, and this technology, although still relatively expensive, can provide both exceptional sensitivity and high specificity for the analysis. Benowitz (1996) has reviewed the trade-offs in terms of relative sensitivities, specificity and cost for these various techniques.

At this time, cotinine remains the biomarker of choice for assessing the exposure of children to SHS in most cases. Several other markers have been examined such as hemoglobin adducts of aromatic amines or PAH-albumin adducts, but they lack specificity as markers for tobacco smoke. However, another highly specific marker of interest is the tobacco-specific nitrosamine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), which is derived from nicotine and which combines a specificity similar to nicotine or cotinine for exposure to SHS with additional relevancy as a presumed carcinogen (Hecht, 1998; U.S. Surgeon General, 2006). NNAL has been measured in urine samples from infants and children exposed to SHS (Hecht et al., 2001, 2006), but the concentrations of this marker are quite low, so current analyses are still somewhat difficult and the sensitivity of NNAL as an SHS exposure marker is less than that of cotinine.

Biomarker Measurements in Medically At-Risk Children

In general, the approaches for estimating SHS exposure in medically at-risk children should correspond to those used for healthy children, but there are essentially no data specifically addressing this point. Since exposure to SHS is known to increase the prevalence of asthma in children (U.S. Surgeon General, 2006), the exposure of asthmatic children has been determined in many studies, and the approaches used for assessing biomarkers or personal exposure monitoring have been the same as those used for a general population. Cotinine is the most common exposure biomarker, and as a nicotine metabolite, its concentration could be influenced by alterations in nicotine metabolism that has been recently reviewed (Hukkanen, Jacob, & Benowitz, 2005). Nicotine is metabolized to cotinine primarily by the action of aldehyde oxidase and the P_{450} enzyme CYP2A6, predominately in the liver. Cotinine may be further metabolized to 3′-hydroxycotinine, also by CYP2A6, and nicotine, cotinine and hydroxycotinine all form glucuronides by UDP-glucuronsyltransferase activity. Nicotine may also be partially converted to its *N*-oxide by action of a flavin monooxygenase. Thus, a disease process or drug therapy that influenced these enzyme activities might affect the conversion of nicotine to cotinine and thus potentially interfere with the estimated exposure measurement. CYP2A6 is the most likely enzyme to be influential in this regard, although other P_{450} MFO enzymes may also be involved in nicotine metabolism.

Hukkanen et al. (2005) note that certain diseases have been shown to affect CYP2A6 activities.

For example, hepatitis A reduces coumarin metabolism (a marker of CYP2A6 activity), whereas CYP2A6 expression may be increased in liver areas adjacent to hepatocellular carcinoma. Whether such changes could influence cotinine metabolism and exposure assessments in a medically compromised child is unknown. Similarly, drugs known or suspected of influencing CYP2A6 activity such as rifampicin or phenobarbital might influence cotinine formation. Several drugs are known to influence CYP2A6 activity in vitro, but at this time only methoxsalen and tranylcypromine have been demonstrated to inhibit the metabolism of nicotine in people (Hukkanen et al., 2005). Drug regimens also might directly affect biomarker measurements in some cases. For example, pemoline, a CNS stimulant that may be used in children, was found to be capable of potential interference even in a highly-specific LC/MS/MS analysis of cotinine in serum (Bernert et al., 1997). An even greater risk of analytical interference by therapeutic drugs might be expected for less specific cotinine assays such as immunoassays. However, although medically compromised children—especially those with reduced lung function—might be expected to be at potentially greater risk from exposure to SHS, there remains little that is known at present of the extent to which disease processes or drug therapy might influence either the assessment of SHS exposure in such children or its impact.

Interview Measures of Exposure

Because they have high face validity, are noninvasive, and are relatively inexpensive to administer, interviews and questionnaires are frequently used to assess the exposure of children and other nonsmokers to SHS. These measures include a wide variety of questions, ranging from whether a child shares a home with a smoker to estimates of how long a child may have inhaled SHS to estimates of how many cigarettes were smoked in a room or car when the child was present.

Different from biomarkers of exposure, interview measures cannot confirm biological exposure but provide indirect evidence that exposure very likely occurred because a child was in the same room with someone who smoked or lives in an apartment in which parents smoke when the child is absent. For interview questions to provide valid proxy measures of exposure, the respondent has to have noticed that smoking occurred, must remember that a child was present, and be able to provide accurate estimates of frequency or duration. Matt et al. (1999, 2000) have shown that parents are often able to do so and that well-constructed interview questions yield quantitative parent reports of exposure that account for 20–40% of the variance in biomarkers of exposure.

While biomarkers of SHS exposure confirm that SHS pollutants have crossed over into the body of a child during a certain period of time, they tell little about how and where past exposure occurred or what can be done to reduce future exposure. In the proposed ecological measurement approach of SHS exposure, the primary role of interview measures is to provide complementary, unique information about the sources, time profile, and locations of exposure. This information can be provided through interview questions asking about smoking behavior in the social network of a child, the activity pattern over time and in different locations, and when a child was in proximity of a smoker or contaminated media. This information can provide the foundation for behavioral assessments and interventions to reduce and prevent future exposure.

Why? Factors that Contribute to Tobacco use and SHS Exposure in the Environment of a Child

From the perspective of an ecological measurement approach, the assessment of SHS exposure should also include a careful examination of the factors that contribute and maintain tobacco use and SHS exposure in the environment of a child. Following the Behavioral Ecological Model, such factors include smoking history of parents, family background, attitudes, family policies and rules, and community standards that affect smoking behavior in public and private spaces. Many states in the US and countries worldwide have established tobacco control policies that restrict smoking in public spaces, such as the workplace, restaurants, hospitals, and schools (Fong et al., 2006a; Fong, Cummings, & Shopland, 2006b;). Yet, public policies to protect children from SHS tend to exclude smoking in private places such as homes, cars, and family child care homes that offer child care in private residences (Moon, Biliter, & Croskell, 2001). Many nonsmoker and smoker parents have voluntarily established rules about where and when smoking is allowed (e.g., patio; when children are absent; Borland et al. (2006a,b)). Yet, these home policies may not be fully protective because SHS can quickly spread to neighboring rooms and linger long after a cigarettes as been smoked, limiting the impact of such rules for protecting children from SHS in these microenvironments (Matt et al., 2004).

Knowing who smokes, when and where smoking takes place, what is contaminated with SHS, how exposure takes place, how much the child is exposed, and why tobacco is used in the environment of a child, interventions can be designed to protect a child from SHS. Based on the proposed ecologic model, interventions can take place at the individual, family, community, or population levels and can target physical and social environments. At the population and community levels, such intervention include mass-media-based health education campaigns, local ordinances that ban smoking on playgrounds, and smoke-free housing initiatives that aim at changing individual behavior, community norms, and the physical environment. At the individual and family levels, this may involve advice given by a physician during a well-visit, health education provided by a nurse, smoking cessation interventions, or intensive counseling for SHS reduction (Gehrman & Hovell, 2003).

Conclusions

Even though SHS exposure has been a major public health concern for more than 20 years (U.S. Surgeon General, 1986), very little is currently known about its consequences for medically at-risk youth. To close this gap, the proposed research plan for the National Children's Study (U.S. Department of Health and Human Services, 2007) could play a critical role. Beginning in 2009, this study will follow 100,000 children throughout the US from before birth to age 21 to better understand how environmental influences and genetic constitution interact to affect child and adult health. The collection of biomarker and interview data could provide a rich and unprecedented source of data to explore the health consequences of SHS exposure in children in general and in health-compromised children in particular. To be successful, such research must be transdisciplinary, involving basic, clinical, and population scientists. In the following, we briefly outline six major research priority areas that deserve special attention to fill existing gaps and to expand our understanding of the role of SHS exposure in this important population.

Orientation Toward Tobacco Smoke Pollution of Environments Relevant to Medically At-risk Children

Further work on the identification of primary SHS exposure locations and sources of children should be conducted. For example, the contribution from smoking in the home relative to the automobile, childcare facilities or playgrounds to the exposure of infants and children should be better defined. This also includes a better understanding of the relative contribution of parents, friends, neighbors, and childcare personnel and the role of different media in a child's environment that might be polluted with SHS contaminants. This research could lead to better understanding the unique pollution and exposure profiles and exposure dosages in the environments of medically at-risk children.

Interaction of Child and Environment

SHS exposure results from the interaction of an individual with pollutants in the environment. Medical conditions and their treatments often impose restrictions on a child as well the environment in which care is provided. Little is understood how these conditions may exacerbate or attenuate the short and long-term consequences of passive smoke exposure. For instance, a better understanding is needed of the respiratory and metabolic differences in processing tobacco smoke toxins among newborns, infants, and children compared to adults and how this is affected by different medical conditions. Similarly, the role that such differences may play in affecting both exposure assessments and disease risks needs further investigations.

Biomarkers and Reported Measures of Exposure

SHS is a complex and dynamic mixture of a large number of toxins. Further development and validation of biomarkers of exposure to different SHS constituents and their temporal and spatial variability are warranted. More work is required on the stability and reliability of biomarker measurements as indicators of integrated SHS exposure over time. In addition, a better understanding of the potential effect of specific disease processes or treatments on current biomarker measurements is needed. More information is needed combining both interview and biomarker data on the specific exposure levels of children with particular health issues, especially children with respiratory diseases of all types.

Development Effects

Healthy and medically at-risk children are at increased risk of SHS-induced illnesses because important organ systems are not yet fully developed or may be especially stressed because of a medical condition. Additional work should be devoted to the association between SHS exposure and neurodevelopmental effects in children. A better understanding is needed using appropriate exposure indices of the role chronic SHS exposure plays

in cognitive, behavioral, and medical problems. Large national studies such as the NHANES and the National Children's Study (NCS) might be especially useful in this regard for adding to our understanding of biomarkers of exposure as well as the social contingencies operating in the environments of children homes that result in SHS exposure. Another example is the role of SHS exposure in promoting early cardiovascular changes potentially contributing to later disease. The association between SHS exposure and potential cardiovascular markers such as lipoprotein concentrations, prostaglandin production, and vitamin levels in young children should be further investigated.

Co-occurrence of Risk Factors

SHS exposure should be viewed in the context of other risk factors as their effects on health outcomes are likely to combine and interact. Future research is needed to examine the co-occurrence of SHS exposure with the exposure to other environmental pollutants, early smoking initiation, poverty, crime, poor nutrition and exercise, and education. This also includes disparities in tobacco smoke pollution and SHS exposure in communities of color and different socio-economic groups. These factors may not only affect health outcomes but also play a role in designing effective prevention and intervention strategies. Many studies of children to date have involved developed countries, often with relatively low exposure levels. More information is needed on exposure levels among third-world children who may have many additional health limitations and may also be exposed to significantly higher SHS exposures.

Prevention and Intervention

Effective strategies to prevent, reduce, and avoid SHS exposure in medically at-risk populations is of particular interest, providing unique opportunities and challenges. Research and development are needed to design and evaluate new approaches at different levels of the ecological system in which SHS exposure takes place, targeting beliefs, attitudes, and behavior and recognizing the important role of communities that communicate and control social norms about tobacco use and SHS.

We have introduced a measurement framework that conceptualizes the exposure of children to secondhand smoke in the social, biological, and physical environments in which the children live. We argue that SHS exposure in children is the consequence of a causal sequence of processes that originate in tobacco use among members of the social network of a child, including parents, extended family, friends, child care providers, neighbors, and local communities. Tobacco use among these network members leads to short- and long-term pollution of the physical microenvironments in which a child lives, including their home, family car, private child car facilities, and parks. Children may be exposed in these microenvironments directly or indirectly to SHS contaminants, leading to measurable doses of biomarker of these contaminants in the body and setting in motion short- and long-term disease processes.

Comprehensive efforts to reduce SHS exposure require strategies to address smoking, contamination, and exposure at the level of individual children, their families, neighborhoods, and communities in which tobacco use leaves its social, economic, physical, and biological scars. This measurement model draws attention to these multiple opportunities for intervening in an effort to reduce and prevent disease outcomes. Literature reviews by Gehrman and Hovell (2003) and Hovell, Zakarian, Wahlgren, and Matt (2000b) suggest that intensive counseling interventions based on sound behavior change theory have yielded the most promising results at the individual and family levels. As the experiences from California's comprehensive tobacco control program have shown, population-wide reductions in tobacco use can occur over relatively short periods of time (10–20 years), leading to significant changes in tobacco consumption, attitudes, norms, and expectations about tobacco use, a proliferation of local smoke-free ordinances (e.g., beaches, parks, and playgrounds), and a population-wide reduction in SHS exposure (Gilpin *et al*., 2003, Gilpin, Messer, White, & Pierce, 2006).

Because most intensive counseling efforts to reduce children's SHS exposure has taken place as part of scientific efficacy trials, we suspect that clinical services do not yet contribute significantly to the reduction of children's exposure to SHS. However, evidence from efficacy trials is now sufficient to warrant promotion of such services, and the Behavioral Ecological Model suggests that such services might be most influential if provided by allied health (e.g., social service agencies, WIC) as well as medical and dental providers, where cumulative exposure to such counseling might reduce and sustain reductions in children's exposure in their home (Hovell et al., 2002b). Such cumulative exposure to multiple clinical services might be especially important to reduce the risks for medically compromised children who are exposed to SHS in their homes. Coordinated clinical services with respect to SHS exposure control are part of a larger ecological change in culture that might be even

more powerful in establishing new norms for home smoking restrictions and protection of children. These processes demand new research in the translation of efficacy studies as well as translation of epidemiology studies of tobacco avoidant cultures, in order to promote both coordinated clinical services and refined tobacco control cultures. This new generation of field trials of effective interventions for community-wide change in residential exposure requires a comprehensive measurement model built on existing behavioral, environmental, and biological measures of exposure as well as new measures to be developed, including those specifically designed for and validated in at-risk populations (e.g., acute and chronic childhood illnesses) and real-time assessment of the time-course of SHS exposure.

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