Assessing secondhand smoke exposure with reported measures

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ABSTRACT
Non-smokers are exposed to tobacco smoke from the burning cigarette and the exhaled smoke from smokers. In spite of decades of development of approaches to assess secondhand smoke exposure (SHSe), there are still unresolved methodological issues. This manuscript summarises the scientific evidence on the use of SHSe reported measures and their methods, objectives, strengths and limitations; and discusses best practices for assessing behaviour leading to SHSe for lifetime and immediate or current SHSe. Recommendations for advancing measurement science of SHSe are provided. Behavioural measures of SHSe commonly rely on self-reports from children and adults. Most commonly, the methodology includes self, proxy and interview-based reporting styles using retrospective recall or diary-style reporting formats. The reporting method used will vary based upon the subject of interest, assessment objectives and cultural context. Appropriately implemented, reported measures of SHSe provide an accurate, timely and cost-effective method for assessing exposure time, location and quantity in a wide variety of populations.

INTRODUCTION
In this series of articles, three topic assessments summarising current knowledge about measuring secondhand smoke exposure (SHSe) are presented, covering self-reported measures, environmental measurement biomarkers, and are based on a multidisciplinary expert meeting held in late 2008 at Johns Hopkins University, Baltimore, USA and supported by the Flight Attendant Medical Research Institute (FAMRI). The meeting addressed SHSe assessment approaches to provide uniform reporting styles using retrospective recall or diary-style reporting formats. The reporting method used will vary based upon the subject of interest, assessment objectives and cultural context. Appropriately implemented, reported measures of SHSe provide an accurate, timely and cost-effective method for assessing exposure time, location and quantity in a wide variety of populations. Aspects of the physical and social environments should be considered when developing a measurement tool as both are drivers of smoking and/or SHSe. Within defined microenvironments, the practices relating to production of SHSe, attenuation, or avoidance of smoke should be assessed over time and by microenvironment. Establishing specific time-activity patterns for the individual in question is important for accurate identification of times and locations of potential exposure. For example, as health-compromised children are likely to spend more time indoors than adults or healthy children, they may be at risk for SHSe. Younger children demonstrating higher hand-to-mouth behaviour may also be exposed to higher SHS residue (e.g. ‘third-hand smoke’) on objects and surfaces. Collecting parental smoking history, family background, attitudes, family rules about smoking in the home, and social and community standards will lend insight into determinants of SHSe, which likely contribute to smoking behaviour and to whether smoking takes place in the individual’s microenvironments, in public and private settings.

MODELS OF EXPOSURE
The microenvironmental exposure model has been adopted to frame assessments of behaviour leading to SHSe. A microenvironment is a space with a volume (e.g. a space in a home, office, school) in which the SHS concentration is ‘spatially uniform’ during some specific time interval.1 The SHS concentration in a microenvironment, at a given time, results from the density of smoking in that environment, mediated by factors diluting or otherwise removing smoke from the air.2 SHSe is expressed as the sum of exposures in the multiple microenvironments where a person spends time.3 4

ASSESSING SHSE IN DIFFERENT SETTINGS
Clinical setting studies
Interviews or questionnaires administered in clinical settings assist doctors in identifying patients with SHSe who may benefit from counselling or other interventions. SHSe assessment is most important in children, non-smoking adolescents, young adults and in asthmatic populations or among those with chronic obstructive pulmonary disease. Lifetime exposure assessment in adults is critical for risk assessment for lung cancer and coronary heart disease, especially among high-exposure workers (e.g. flight attendants, bartenders and waiters) and those who have lived with a smoker. SHSe is generally measured by proxy (parent or other adult) for young and school aged; self-report for young children; and adolescents. For
adolescents, the distinction between tobacco use and SHSe from others should be included. Ensuring confidentiality is critical to prevent disclosure of potentially illicit smoking behaviour by teens or their peers to their parents or guardians. SHSe assessment by doctors in a clinical setting, however, is often problematic because clinicians are charged to complete many tasks in a short period. SHSe assessment is not their priority, and they often feel undertrained about intervening in SHSe situations.1,7

Field and epidemiological studies
Assessing SHSe at the population level requires techniques that can be implemented in large samples. Study needs, however, may be addressed with simple, straightforward approaches if the goals are to measure exposure prevalence. Large population-based surveys, such as the National Health and Nutrition Examination Survey (NHANES), the Global Youth Tobacco Survey (G YTS), the International Tobacco Control (ITC) Surveys, the Global Adult Tobacco Survey (GATS) and smaller surveys with more focused populations of interest have assessed SHSe in the home and in public places including entertainment venues. Smoking restrictions and bans at home and in the community for microenvironments (eg, restaurants, bars, stores and outdoor environments) are important measures to collect. For example, the GATS, which has been implemented in more than 15 countries, includes detailed questions related to the rules of smoking inside the home and work. The inclusion of these SHSe questions should be guided by local smoke-free practices of the population of interest. These measures can be useful for smoke-free policies’ implementation and compliance evaluation and thus be related to respondents as contextual variables in multilevel research designs.3

In epidemiological studies, a valid and reliable measurement of SHSe is fundamental to accurately assess disease risk, as inaccurate measurement of a person’s exposure can lead to biased risk estimates2 and is often cited as a major limitation of studies evaluating association between SHSe and disease. Understanding the nature of the misclassification is critical, as systematic measurement errors can bias risk estimates towards or away from the null, while random errors in exposure misclassification can diminish the exposure–risk relationship. In SHSe studies, where the hypothesised association between exposure and disease is anticipated to be modest, imprecise exposure measurement could lead to a conclusion that a real risk, while modest, does not exist (type 2 error). For SHSe in a population where a large number of people could be affected, even a modest disease risk has important public health implications.

When developing the questionnaire, its method of administration and cultural context for assessing SHSe must be considered. The use of culturally competent instruments and methods will help ensure accuracy by increasing the rate, validity and reliability of the responses.7 To maintain reliability and validity, the materials’ language, microenvironments and administration must be contextually appropriate. Translated questionnaires should match the original in language, semantics, idioms, experiences and concepts.2 Questions about exposure to types of tobacco smoke other than cigarettes should be revised as appropriate.

REPORTED METHODS FOR ASSESSING SHSE
Questionnaires are the most common method of assessing SHSe because they are simple to administer and cost effective compared to biological markers and environmental monitoring.2 10 An added advantage is that individuals can report lifetime SHSe as well as usual and current levels. While some studies also include measures of possible determinants of smoking or exposure behaviour, such as education or working outside of the home, these remain under development.1,12

False reporting, or over/under-reporting, are potential limitations of using questionnaires and will vary depending on the cultural context of smoking and SHSe. However, these limitations are not unique to data obtained from questionnaires. For example, if participants switch urine samples to avoid biomarker detection, underascertainment results are similar to false reports of being a non-smoker. Cross-measurement correlations (eg, r ≈ 0.50) show relationships among reported smoking and reported child exposure that are roughly equivalent to ‘objective’ measures of nicotine or cotinine (a metabolite of nicotine used as a biomarker of SHSe), suggesting that no method is error free.10,12

A participant’s ability to recall SHSe episodes, including frequency and duration, may also be questionable.16 Recall accuracy in survey responses can be improved by reducing the timeframe between the discrete event and the length of the recall period16; for example, recall over the last 24 h would be more accurate than over 30 days or 6 months. The assumption, however, that events in the last 24 h represent a respondent’s current experiences may be inaccurate, as that 24-h period may be atypical.5,17 Researchers need to balance between striving to achieve a respondent’s accurate recall and the need to cover a longer period to obtain more representative SHSe estimates. To balance reporting accuracy against research requirements, we recommend a maximum 7-day recall period in a single assessment. However, useful long-term indicators reported with reasonable accuracy can include living with smoking parents, spouse, or other household members.

Another SHSe assessment method is a diary, which can provide participant’s detailed exposure information within specific microenvironments. Diaries reduce the recall burden: as reporting occurs over the past day rather than the past week or month, the respondent knows what should be reported in the diary later that day and will likely notice and record instances of exposure as they occur.18 Direct observation (live or video recorded) with trained observers can increase objectivity, reducing the rate of false reporting. It is likely to provide more valid data due to focused attention by the trained data collector, whether an individual study participant or a study team member. If used together, diary and observational methods can be quite informative for respondents or microenvironments of particular interest or for providing comparison data to estimate measurement error.

ACCURACY OF QUESTIONS ASSESSING SHSE
Questions assessing SHSe should be valid and reliable (ie, accurate). Participants’ answers to SHSe questions are deemed reliable if those answers are replicated or reproduced when the questions are repeated.19 Binary response (ie, yes/no) reliability can be measured using the κ statistic (the observed agreement beyond chance), while correlation coefficients can be used for continuous responses. In general, a κ value > 0.75, 0.40–0.75 and below 0.40 represents excellent, intermediate to good and poor agreement beyond chance, respectively.19 In interpreting Pearson’s correlation coefficient, we refer to reliability as: none (0.0 to ±0.09), low (±0.1 to ±0.29), moderate (±0.3 to ±0.49) and strong (±0.5 to ±1.0). Studies testing the validity of SHSe measurement questions assess the questions’ ability to correctly identify those individuals truly exposed, along with exposure intensity.19 Acceptable ‘gold standards’ are environmental...
measures, including airborne nicotine or respiratory particles, and biomarkers, such as cotinine. The relationship of the environmental measure with the questionnaire items will vary depending on time since peak exposure, level of continuous versus intermittent exposure, air sampling time frame and measured microenvironments.20 21

Respondents may not realise that they have been exposed to SHS. For example, in NHANES 2003–2004, overall self-reported exposure prevalence was underreported as compared to serum cotinine, but the underreporting level depended on the exposure definition.22 For instance, 42% of non-smoking adults had detectable levels of cotinine, while only 5.4% reported exposure at home and 8.5% reported exposure at work. For home exposure measurement, one selected family member reported whether any household member smoked anywhere inside the home. SHSe classification at work was based on each participant’s response to the number of hours per day that he or she could smell tobacco smoke.22 Thus, the specific nature of measures can lead to misclassification in exposure estimates.

To estimate total exposure, the survey respondent should recall and quantify exposure across different locations, length of exposure and its source. Instructing respondents on proper definitions and providing examples of events that qualify, as well as possible SHSe locations, improve recall accuracy.23 Surveys intending to capture exposure intensity, estimated time of exposure, number of smokers generating SHS and proximity to smokers should include items addressing these factors specifically.24

A comprehensive Medline literature search of English-language journals was conducted, covering January 1966 to 13 July 2010, and using the search terms ‘tobacco smoke pollution’ (MESH terms) AND questionnaire AND (valid* OR Reproduc* OR reliable*) for studies among humans. Of the 102 articles examined, 3 presented relevant information on questionnaire reliability; 5 others were identified through reference lists of included articles and reviews. A total of 45 validation studies were identified (air nicotine or particulate matter concentrations (15), measurements of hair nicotine (4) and cotinine (28)).

Reliability of questions assessing SHSe

Adults can reliably answer questions regarding SHSe (yes/no) from their parents during childhood (κ=0.82), their spouse, household members, and if they are living with a spouse or mother who smokes (table 1).25–27 However, reliability sharply decreases for exposure from father, siblings, own children and other household members.27 This includes responses related to years of SHSe to maternal (r=0.69–0.76), paternal (r=0.75) and other household members’ smoking (r=0.63) during childhood, as well as for years of exposure by their spouse (r=0.95) or other household members (r=0.78) during adulthood.28 Correlation coefficients ranged from 0.18 to 0.55 for hours per day,26–20 or weeks.25

Studies of the assessment of children’s SHSe have reported good reliability using parents’ self-reported smoking behaviour including amount, type of day (weekday/weekend) and place of exposure (table 2).26 29 Families of children with asthma (n=91) kept a 2-week diary recording cigarettes per day (CPD) smoked in the home, by type of day, at baseline and 2 months later.16 Overall, reliability was high for child CPD exposed at home per week or during a typical workday, by all sources (r=0.81) and by the parents (r=0.75). Reliability was lower for parental CPD exposure at home on a typical non-workday (r=0.63). In another study, high test–retest correlations were obtained for questions about CPD smoked by the mother, total, or at home, either on weekdays or weekends (r=0.95), when again repeatedly at baseline, 3-month and 6-month interview.29 Mothers of children with asthma gave highly reliable answers regarding CPD smoked while the child was present in the room or car with smokers (r=0.69).30

The reliability of participants’ answers to questions about their childhood and adulthood SHSe was compared with surrogates’ responses. Surrogates included parents, siblings, spouses, other household members and coworkers. Good agreement was reported between the participants and surrogate responses in nearly all SHSe measures, including presence and number of smokers at home (r=0.67), number of years exposed (r=0.89) and an index of SHSe (severity × years of exposure) (r=0.74).21

Validity of questions assessing SHSe using environmental measurements

The number of smokers or of cigarettes to which non-smokers are exposed may be valid indicators of SHSe intensity (supplementary table S1). Several studies evaluating the correlation between number of cigarettes smoked in the home and air nicotine concentration also found strong correlation coefficients.29 32 33 Two studies of families of children with asthma, however, reported low or no correlation; both used air nicotine passive monitors.18 34 In a study of 254 non-smoking pregnant women wearing air nicotine passive monitors for 1 week, low to moderate correlations were found between total number of smokers or cigarettes to which the women reported exposure in the homes (0.34–0.35) and at work (r=0.16–0.17).35 Strong correlation coefficients were reported for the number of smokers present or to whom the person was exposed at work (r=0.61–0.67),36–38 and the number of cigarettes smoked in a restaurant (r=0.78) (supplementary table S2).

Two studies validated questions about SHSe intensity among children (supplementary table S3).18 20 Correlations between air nicotine measurements and the reported number of cigarettes to which the child (6–17 years old) was exposed and CPD smoked in the house ranged from 0.07 to 0.43.36 Responses from mothers of young children were moderately to strongly correlated with air nicotine levels measured with an active monitor.29 Strong, nearly identical correlation coefficients were found between the number of cigarettes to which the child was exposed at home from the mother (r=0.50–0.64) and the number of cigarettes smoked inside the home per week by the mother (r=0.65–0.84).29

Several studies have validated questions assessing the duration (hours or minutes) of SHSe using air nicotine concentrations (supplementary tables S1–S3).34 35 37 39 40 In these studies, the correlations were found non-existent to moderate. As questions about exposure location became more specific, the correlations became weaker. Specifically asking about the number of hours a child is exposed at home yielded low to moderate correlation coefficients in two other studies (r=0.25–0.36).34 40

Coghlin evaluated the validity of a 7-day diary and a questionnaire against air nicotine among 19 non-smoking adults.24 The strongest correlations (r=0.91–0.95) were found with an index based on information about the hours of exposure, number of smokers and their proximity to the participant (supplementary table S4).

Validity of questions assessing SHSe using biomarkers

Questions regarding children’s SHSe and exposure intensity have been validated with the presence of nicotine and cotinine in hair (supplementary table S5).41–44 Al-Delaimy et al found that hair nicotine levels in children increased 10.5% per mother’s

Review
Table 1 Reliability of questions assessing secondhand smoke exposure (SHSe) among adults

<table>
<thead>
<tr>
<th>Reference population</th>
<th>Assessment</th>
<th>Exposure time</th>
<th>Exposure source</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>SHSe status (yes/no)</td>
<td>SHSe status</td>
<td>Pregnancy</td>
<td>Mother</td>
<td>$k=0.73^{***}$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Childhood</td>
<td>Mother</td>
<td>$k=0.87^{***}$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>$k=0.85^{***}$</td>
</tr>
<tr>
<td>Coultas et al, 1989\textsuperscript{25}: 149 participants aged 21—79 years asked 4—6 months apart asked if the source had smoked daily for $\geq 6$ months</td>
<td>SHSe status</td>
<td>Childhood</td>
<td>Parents</td>
<td>93.6%, $k=0.82$; SE=0.095</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>All household members</td>
<td>82.7%, $k=0.60$; SE=0.090</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Spouse</td>
<td>83.6%, $k=0.67$; SE=0.095</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>All household members</td>
<td>80.0%, $k=0.59$; SE=0.093</td>
</tr>
<tr>
<td>Browson et al, 1993\textsuperscript{27}: 37 cases and 73 controls asked 1.7—1.9 years apart. All non-smoking women participants of a larger lung cancer study in Missouri.</td>
<td>SHSe status</td>
<td>Childhood</td>
<td>Adolescence</td>
<td>$k=0.66$; SE=0.08 (females: $k=0.61$; SE=0.11)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wife</td>
<td>N=53; $k=0.89$; SE=0.064</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Husband</td>
<td>N=61; $k=0.89$; SE=0.059</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Children</td>
<td>$k=0.24$; SE=0.106</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mother</td>
<td>$k=0.76$; SE=0.103</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>$k=0.44$; SE=0.077</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sibling</td>
<td>$k=0.57$; SE=0.117</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other (relatives)</td>
<td>$k=0.16$; SE=0.137</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other (non-relatives)</td>
<td>$k=0.02$; SE=0.093</td>
</tr>
<tr>
<td>Living with a smoker (yes/no)</td>
<td>Ever living in a house</td>
<td>Lifetime</td>
<td>Any</td>
<td>$r=0.45$; 95% CI 0.29 to 0.58 (males: $r=0.46$; 95% CI 0.24 to 0.64)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wife</td>
<td>N=56; $r=0.25$; 95% CI –0.01 to 0.48</td>
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<tr>
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<td></td>
<td>Husband</td>
<td>N=36; $r=0.20$; 95% CI –0.14 to 0.50</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Parent</td>
<td>N=40; $r=0.48$ 95% CI 0.20 to 0.69</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Mother</td>
<td>N=8; $r=0.69$; 95% CI –0.03 to 0.94</td>
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<tr>
<td></td>
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<td></td>
<td>Father</td>
<td>N=32; $r=0.46$ 95% CI 0.13 to 0.70</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Other (relatives)</td>
<td>N=17; $r=0.59$; 95% CI 0.15 to 0.83</td>
</tr>
<tr>
<td>Browson et al, 1993\textsuperscript{27}: 37 cases and 73 controls asked 1.7—1.9 years apart. All non-smoking women participants of a larger lung cancer study in Missouri.</td>
<td>Years of SHSe</td>
<td>Lifetime</td>
<td>Any</td>
<td>$r=0.45$; 95% CI 0.29 to 0.58 (males: $r=0.46$; 95% CI 0.24 to 0.64)</td>
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<tr>
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<td>Wife</td>
<td>N=56; $r=0.25$; 95% CI –0.01 to 0.48</td>
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<td>Husband</td>
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<td>Parent</td>
<td>N=40; $r=0.48$ 95% CI 0.20 to 0.69</td>
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<tr>
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<td></td>
<td>Mother</td>
<td>N=8; $r=0.69$; 95% CI –0.03 to 0.94</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>N=32; $r=0.46$ 95% CI 0.13 to 0.70</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Other (relatives)</td>
<td>N=17; $r=0.59$; 95% CI 0.15 to 0.83</td>
</tr>
<tr>
<td>Pron et al, 1988\textsuperscript{27}: 117 controls (63 females and 54 males) in a case-control study of lung cancer in Toronto asked 6 months apart</td>
<td>Years of SHSe</td>
<td>Childhood</td>
<td>Mother</td>
<td>N=33; $r=0.76$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>N=57; $r=0.75$</td>
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<tr>
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<td></td>
<td>Others</td>
<td>N=26; $r=0.63$</td>
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<td></td>
<td></td>
<td>Spouse</td>
<td>N=40; $r=0.95$</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Others</td>
<td>N=40; $r=0.95$</td>
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<td>Other household members</td>
<td>N=67; $r=0.78$</td>
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<td></td>
<td></td>
<td></td>
<td>Child</td>
<td>N=31; $r=0.18$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>N=55; $r=0.54$</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Others</td>
<td>N=20; $r=0.51$</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Other household members</td>
<td>N=58; $r=0.54$</td>
</tr>
<tr>
<td>Pron et al, 1988\textsuperscript{27}: 117 controls (63 females and 54 males) in a case-control study of lung cancer in Toronto asked 6 months apart</td>
<td>Years of SHSe sharing the home with smoker(s)</td>
<td>Adolescence</td>
<td>Father</td>
<td>N=48; $k=0.22^{*}$</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>Spouse</td>
<td>N=79; $k=0.04^{*}$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Spouse</td>
<td>N=64; $k=0.04^{*}$</td>
</tr>
<tr>
<td>Intensity of exposure</td>
<td>Categorical cigarettes per day (&lt;1, 1 –&gt; 1 pack/day, or unknown)</td>
<td>Childhood</td>
<td>Father</td>
<td>N=48; $k=0.22^{*}$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Child</td>
<td>N=31; $r=0.18$</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Father</td>
<td>N=55; $r=0.54$</td>
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<td></td>
<td></td>
<td></td>
<td>Others</td>
<td>N=20; $r=0.51$</td>
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<td></td>
<td></td>
<td></td>
<td>Other household members</td>
<td>N=58; $r=0.54$</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Spouse</td>
<td>N=79; $k=0.04^{*}$</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Spouse</td>
<td>N=64; $k=0.04^{*}$</td>
</tr>
<tr>
<td>Pron et al, 1988\textsuperscript{27}: 117 controls (63 females and 54 males) in a case-control study of lung cancer in Toronto asked 6 months apart</td>
<td>Number of smokers living in the home</td>
<td>Adolescence</td>
<td>No. of smokers living in the home</td>
<td>$k=0.55$; SE=0.071 (females: $k=0.52$; SE=0.1) (males: $k=0.57$; SE=0.1)</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01; ***p<0.001.

\(r\), correlation coefficient.

\(k\), kappa; \(r\), correlation coefficient.

\(\text{SE}\), standard error.

\(\text{CI}\), confidence interval.

\(\text{Pack}\)-years of SHSe

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**Table 2** Reliability of questions assessing current secondhand smoke exposure (SHSe) among children

<table>
<thead>
<tr>
<th>Reference population</th>
<th>Assessment</th>
<th>Exposure source</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zakarian et al, 2004&lt;sup&gt;26&lt;/sup&gt;: mothers with children aged ≤ 4 years recruited in a randomised controlled trial. The time between questions was immediate and repeated at baseline, 3-month and 6-month follow-up.</td>
<td>CPD: asked to report their smoking on typical days during the past 7 days</td>
<td>Mother</td>
<td>Weekday: r = 0.97 – 0.98; weekend: r = 0.95 – 0.99</td>
</tr>
<tr>
<td>Emerson et al, 1995&lt;sup&gt;30&lt;/sup&gt;: 91 families with an asthmatic child 6–17 years old asked 2 months apart</td>
<td>CPD smoked at home: diary (2-week period)</td>
<td>Parents</td>
<td>Workday: r = 0.88; non-workday: r = 0.60</td>
</tr>
<tr>
<td>Zakarian et al, 2004&lt;sup&gt;26&lt;/sup&gt;: mothers with children ≤ 4 years recruited in a randomised controlled trial. The time between questions was immediate and repeated at baseline, 3-month and 6-month follow-up.</td>
<td>CPD smoked at home: in the past 7 days</td>
<td>Mother</td>
<td>r = 0.97 – 0.98</td>
</tr>
<tr>
<td>Emerson et al, 1995&lt;sup&gt;30&lt;/sup&gt;: 91 families with an asthmatic child 6–17 years old asked 2 months apart</td>
<td>Average CPD to which the child is exposed at home: diary (2-week period)</td>
<td>Not specified</td>
<td>r = 0.84</td>
</tr>
<tr>
<td>Matt et al, 2007&lt;sup&gt;22&lt;/sup&gt;: 22 mothers of children &lt; 2.5 years old from an ethnically diverse sample of low-income, low-education families asked 2 months apart</td>
<td>Weekly no. of cigarettes smoked at home: in the past 7 days</td>
<td>Father</td>
<td>r = 0.91</td>
</tr>
<tr>
<td>Emerson et al, 1995&lt;sup&gt;30&lt;/sup&gt;: 91 families with an asthmatic child 6–17 years old asked 2 months apart</td>
<td>Weekly no. of cigarettes smoked at home: diary (2-week period)</td>
<td>Parents</td>
<td>Workday: r = 0.82; non-workday: r = 0.63</td>
</tr>
<tr>
<td>Matt et al, 2007&lt;sup&gt;22&lt;/sup&gt;: 22 mothers of children &lt; 2.5 years old from an ethnically diverse sample of low-income, low-education families asked 2 months apart</td>
<td>Weekly no. of cigarettes to which the child is exposed at home: diary (2-week period)</td>
<td>All smokers</td>
<td>r = 0.81</td>
</tr>
<tr>
<td>Hovell et al, 2002&lt;sup&gt;23&lt;/sup&gt;: 204 children with asthma 3–17 years old asked 5 days apart</td>
<td>Weekly no. of cigarettes to which the child is exposed at home: diary (2-week period)</td>
<td>Other</td>
<td>r = 0.88</td>
</tr>
<tr>
<td>Zakarian et al, 2004&lt;sup&gt;26&lt;/sup&gt;: mothers with children aged ≤ 4 years recruited in a randomised controlled trial. The time between questions was immediate and repeated at baseline, 3-month and 6-month follow-up.</td>
<td>Weekly no. of cigarettes to which the child is exposed at home: in the past 7 days</td>
<td>Mother</td>
<td>r = 0.90 – 0.93</td>
</tr>
</tbody>
</table>

*p <0.05; **p <0.01; ***p <0.001.
†Percentage agreement.

CPD, cigarettes per day; κ, kappa; r, correlation coefficient.

CPD smoked inside the house. Small younger increases were reported for smoking by the father or other adults. Hair nicotine levels in 112 children were highly correlated with CPD smoked by parents (r = 0.68) or by household adults and visitors (r = 0.69) inside the house. High correlations were also reported between hair nicotine concentration and the number of smokers living in the house. In a study of 72 young children, CPD to which a child was exposed was strongly correlated (r = 0.64) with hair nicotine levels. Woodruff et al., however, found a correlation of approximately 0.2 between the total number of cigarettes smoked in a child’s presence in the past month and hair nicotine levels. One possibility for this lack of correlation is poor recall of the number of cigarettes smoked in the child’s presence, as well as length of time to recall (1 month). In a multicountry study, hair nicotine levels among children and non-smoking women increased with increases in number of smokers living in the household, father’s CPD and if smoking was permitted in the house. Additionally, children’s hair nicotine increased if their mother smoked and her CPD, as well as with number of smokers sharing the child’s bedroom. A study among non-smoking bar and restaurant workers showed higher hair nicotine levels with more sources of SHSe and higher values for those working in a bar. Between 1989 and 2009, 23 studies reported on urine cotinine and SHSe among adults and children (supplementary table S6). Four of these studies validated SHSe metrics in adults, with all but one study restricted to non-smokers. Most of the studies found a low to strong correlation between SHSe and urine cotinine-to-creatinine ratio (r = 0.18 – 0.57). Correlation coefficients were strongest when the SHSe question was more specific. Cummings et al found no-to-low correlation between urine cotinine and SHSe using various metrics, including total minutes of SHSe (r = 0.18), size, and ventilation properties of exposure location (r = 0.24 and 0.25, respectively), and total number of SHSe (r = 0.23). O’Connor et al. investigated SHSe in pregnant women by asking if they had SHSe for at least 1 h during the week they were monitored; the investigators found poor agreement with urine cotinine concentration (κ = 0.08). Of the 18 studies validating parents’ report of SHSe in children using urine cotinine, exposure measurements were highly correlated with questions that were more specific and captured exposure intensity (supplementary table S6). Poor agreement was reported for children’s SHSe in the home (κ = 0.35), in the same room as a smoker (κ = 0.11) and SHSe in the past 2 days (κ = 0.05 – 0.11). Willers et al, using an intensity scale based upon household members’ smoking status, found strong correlation with urine cotinine (κ = 0.61). Another study collecting information on the number of cigarettes smoked inside the house, as well as number of household smokers, also showed a strong correlation (κ = 0.59). CPD and number of smokers in the household moderate to strong correlations with cotinine measurements in several studies. A study validated questions among infant’s NNAL (4-[methylaminomethyl]-1-[3-pyridyl]-1-butanol) levels in urine. The correlation between cigarettes smoked, in the home and car, and NNAL concentrations was low.

Other methods of validation of SHSe questions include using cotinine in saliva, blood, plasma or serum (supplementary table S7). Studies validating adult’s reported SHSe questions...
Table 3  Recommended questions for studies assessing secondhand smoke exposure (SHSe)

<table>
<thead>
<tr>
<th>Question</th>
<th>Location</th>
<th>Source</th>
<th>Reliable*</th>
<th>Validation air*</th>
<th>Validation biomarker*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lifetime</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of years living with a smoker</td>
<td>Home</td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adulthood</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Living with a regular smoker (yes/no)</td>
<td>Home</td>
<td>Wife</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Husband</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of years sharing the home with smoker(s)</td>
<td>Home</td>
<td>Spouse</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other HM</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of pack-years of SHSe (number of cigarette packs smoked per day by the source × number of years the source has smoked)</td>
<td>Total</td>
<td>Parents</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>All HM</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of cigarettes smoked</td>
<td>Home</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Saliva cotinine</td>
</tr>
<tr>
<td></td>
<td>Work</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>No. of smokers</td>
<td>Work</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Saliva cotinine</td>
</tr>
<tr>
<td>No. of hours of SHSe</td>
<td>Work</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>No. of hours of exposure × no. of smokers × proximity to the participant</td>
<td>Total</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Nicotine/ particulate matter</td>
</tr>
<tr>
<td>Childhood (past)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SHSe during childhood (yes/no)</td>
<td>Total</td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Father</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SHSe while mother was pregnant (yes/no)</td>
<td>Total</td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Father</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other HM</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of hours per day of SHSe</td>
<td>Total</td>
<td>Father</td>
<td>Yes</td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other HM</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of pack-years of SHSe</td>
<td>Total</td>
<td>Parents</td>
<td>Yes</td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All HM</td>
<td>Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnant women (third trimester)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hours of woman’s SHSe at home</td>
<td>Home</td>
<td>All HM</td>
<td></td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td>Cigarettes per day smoked in their presence</td>
<td>Home</td>
<td>All HM</td>
<td></td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td></td>
<td>Home or work</td>
<td>All HM and coworkers</td>
<td></td>
<td></td>
<td>Cord blood cotinine</td>
</tr>
<tr>
<td>Childhood (current)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking status of parent</td>
<td>Total</td>
<td>Mother</td>
<td></td>
<td></td>
<td>Saliva cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Father</td>
<td></td>
<td></td>
<td>Saliva cotinine</td>
</tr>
<tr>
<td>Hours of child’s SHSe at home</td>
<td>Home</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Nicotine†</td>
</tr>
<tr>
<td>Cigarettes per day smoked</td>
<td>Total</td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td>Plasma cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All HM</td>
<td></td>
<td></td>
<td>Plasma cotinine</td>
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<tr>
<td>Cigarettes per day smoked in the home</td>
<td>Home</td>
<td>Mother</td>
<td>Yes</td>
<td></td>
<td>Urine cotinine/ hair nicotine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parents</td>
<td>Yes</td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All HM</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All HM and visitors</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hair</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>Cigarettes per day smoked in the presence of the child</td>
<td>Home</td>
<td>All smokers</td>
<td>Yes</td>
<td></td>
<td>Urine cotinine†</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Har nicotine</td>
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<tr>
<td>No. of cigarettes smoked</td>
<td>Total</td>
<td>Mother</td>
<td></td>
<td></td>
<td>Nicotine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>No. of cigarettes smoked in the home</td>
<td>Home</td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Nicotine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mother</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>No. of cigarettes smoked in the presence of the child</td>
<td>Total</td>
<td>Mother</td>
<td></td>
<td></td>
<td>Nicotine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>All smokers</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mother</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parents</td>
<td>Yes</td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
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<td>Car</td>
<td>Yes</td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td>Weekly no. of cigarettes smoked</td>
<td>Total</td>
<td>Mother</td>
<td></td>
<td></td>
<td>Urine cotinine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Father</td>
<td>Yes</td>
<td></td>
<td>Nicotine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Parents</td>
<td>Yes</td>
<td></td>
<td>Nicotine</td>
</tr>
</tbody>
</table>

Continued
with saliva cotinine concentrations found moderate correlations with questions about total hours ($r=0.34-0.45$) or minutes ($r=0.56$) of SHSe. $^{37} 55$ 56 However, daily SHSe poorly agreed ($k=0.15$) with saliva cotinine. $^{55}$ Validation studies of children’s SHSe showed intermediate to strong correlations saliva cotinine levels with dichotomous reports of smoking in the home ($k=0.44-0.47$), and maternal and paternal smoking status ($r=0.51-0.56$). $^{13}$ Paternal, maternal and total CPD were moderately to strongly correlated with children’s plasma cotinine levels ($r=0.40-0.59$). $^{15}$ Data from NHANES III obtained from 2516 children aged 4–16 years showed a strong correlation ($r=0.57-0.73$) between number of household smokers and serum cotinine. $^{14}$ A study among pregnant women in their third trimester found strong correlations of cord blood cotinine levels with SHSe duration ($r=0.52$) and CPD exposed ($r=0.52$). $^{57}$

**CONCLUSIONS**

Research on the health effects of SHSe has been conducted for over 20 years in the USA. $^{58}$ 59 A variety of instruments, although limited in their accuracy, have been developed and applied; they have proven useful for conducting aetiological studies and tracking SHSe of populations. Extensive experience with questionnaires allows us to (1) offer recommendations for over three decades, and exposure surveillance has been in place for over 20 years in the USA. $^{58}$ 59. A variety of instruments, although limited in their accuracy, have been developed and applied; they have proven useful for conducting aetiological studies and tracking SHSe of populations. Extensive experience with questionnaires allows us to (1) offer recommendations for core questions for SHSe assessment. For example, FAMRI CoE investigators are determining the accuracy of a set core questions, available for investigators at the FAMRI American Academy of Pediatrics Richmond Center of Excellence's website, for the assessment of children’s SHSe.

Acknowledgements The authors would like to thank Nicole Ammerman and Charlotte Gerczak for their technical and editing assistance. The authors would also like to thank Drs Wooel Al-Delaimy, Benjamin J Apleberg, David L Ashley, Neal L Benowitz, John T Bennett, Dana Best, Lara Gundel, Katharine Hammond, Stephen Hecht, Andrew Hyland, Sungroul Kim, Neil Klepeis and James Repace for their participation in the workshop.

Contributors EA-T organised and participated in the expert meeting, drafted and revised the paper. She is guarantor. JLE drafted and revised the paper. KMC, GTF, MFH, JDK, RMcM and JPW participated in the expert meeting, drafted and revised the paper. JMS initiated and organised the expert meeting and revised the draft paper.

Funding This work was supported by grants from the Flight Attendant Medical Research Institute to the Johns Hopkins Center of Excellence; the University of California, San Francisco Bland Lane Center of Excellence; and the American Academy of Pediatrics Julius B Richmond Center of Excellence. The funding organisation had no role in the preparation of the manuscripts.

Competing interests KMC has received payment as an expert witness in litigation against the tobacco industry.

Provenance and peer review: Not commissioned; externally peer reviewed.

Data sharing statement: Manuscript does not contain original data.

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**Table 3 Continued**

<table>
<thead>
<tr>
<th>Question</th>
<th>Location</th>
<th>Source</th>
<th>Reliable*</th>
<th>Validation air*</th>
<th>Validation biomarker*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weekly no. of cigarettes smoked in the home in the presence of the child</td>
<td>Home</td>
<td>Mother</td>
<td>Yes</td>
<td>Hair nicotine/urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
</tr>
<tr>
<td>Weekly no. of cigarettes smoked in the presence of the child</td>
<td>Total</td>
<td>All smokers</td>
<td>Yes</td>
<td>Hair nicotine/urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
</tr>
<tr>
<td>No. of smokers</td>
<td>Total</td>
<td>All smokers</td>
<td>Yes</td>
<td>Hair nicotine/urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
</tr>
<tr>
<td>Cigarette butt counts in 24 h</td>
<td>Home</td>
<td>All HM and visitors</td>
<td>Urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
</tr>
<tr>
<td>Cigarette butt counts × hours spent in living room</td>
<td>Home</td>
<td>All HM</td>
<td>Urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
<td>Hair nicotine/urine cotinine</td>
</tr>
</tbody>
</table>

* $k \geq 0.75$ or correlation coefficient ($r$) $\geq 0.50$.
†Reported by the smoking parent.
$\beta$, hair nicotine; HM, household members.
REFERENCES


64. Environ Health Perspect 1999;107:1034–42.


72. Environ Health Perspect 1999;107:1161–70.


