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Mini-review

Methods for Measuring Exposure to Environmental Tobacco Smoke (ETS)

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ABSTRACT : An important and somewhat under-emphasized issue in assessing the risk potentially associated with exposure to ETS is the reliability and objectivity of the methods used to measure the actual level of exposure. Objectivity of the science is crucial in this context since this topic tends to elicit strong social, emotional, and political responses among the general public and the scientific community alike. This mini-review compares the strengths and limitations of various methods used in determining ETS exposure in scientific investigations. Methods reviewed include: questionnaire, area monitoring, bio-monitoring, and personal monitoring. In particular, results of several recent studies (conducted in the United States, Europe, and the Far East) using personal monitors in combination with questionnaire and bio-monitoring, which contribute to a more reliable, objective and realistic estimates of a person's actual exposure to ETS, are discussed.

Key words **: environmental tobacco smoke (ETS), exposure measurement**

As a component of indoor air pollution, ETS is an unpredictable and constantly changing mixture of chemicals whose complexity and composition may be affected by the kind and frequency of cigarettes being smoked, human activities, and chemical and physical transformation processes. In real life ETS is highly diluted by the ambient air and most ETS components are present at extremely low concentrations. Hence, the measurements of actual levels of exposure, which are determined by the concentration of ETS and the duration of exposure, are difficult (Guerin *et al.*, 1992; Law and Hackshaw, 1996; Witschi *et al.*, 1995; Armitage *et al.*, 1997).

Reliable and objective data of actual levels of ETS exposure are rare. Many studies have investigated ETS as though the mixture was a single agent. Individual components of the mixture or indicators of source strength were used as indices of exposures, with the assumption that the surrogate measures of particles, volatile components, or supposedly tobacco-specific chemicals could accurately reflect the complex ETS mixture in different environments. All published studies lack reliable information about the complex pattern of long term past exposures (Guerin *et al.*, 1992; Law and Hackshaw, 1996; Witschi *et al.*, 1995; Armitage *et al.*, 1997). Some diseases allegedly associated with exposure to ETS, e.g., lung cancer and coronary heart diseases, have long latency periods (Glantz and Parmley, 1991; Aviado, 1996; Raynolds and Fontham, 1995). Because the time interval from initial exposure to a potentially damaging agent to disease development and detection (or symptom onset) is often a decade or longer, there are great uncertainties associated with assessment of long term

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past exposure (Armitage *et al.*, 1997; Rosenbaum *et al.*, 1996; LeVois and Layard, 1994).

Methods for Determining Personal Exposure to ETS

In principle, four methods are used for ETS exposure assessment. Table 1 summarizes some strengths and potential limitations of each of the four methods.

Questionnaires. The questionnaire approach involves asking questions by telephone or personal interview and is used most extensively to provide a relatively simple categorization of ETS exposure. Questionnaire may be asking about past or recent ETS exposures. Questions could be answered by the subject participating in the study or a proxy, depending on whether the subject is alive at the time of the data collection. Basically, questionnaires require subjects to estimate their lifetime exposure to ETS, by asking questions about exposure in different settings throughout life. Sample questions could include: living with a smoker; number of smokers present; number of cigarettes smoked per day; years in marriage to a smoker; the average number of hours per day of exposure to different smokers in the household; whether the

Table 1. Strengths and potential limitations of methods for measuring exposure to ETS

Method	Strengths	Potential Limitations
Questionnaire	• Cost effective • Easy execution • Non-invasive • Detailed information	• Subjective • Recall bias • Intentional falsification hard to check and verify • Misclassification owing to respondents or investigators • Difficult to standardize
Area (stationary) monitoring	• Non-invasive • Objective, integrated measurements • Technical reliability	• Inability to measure time of exposure • Limited to measuring present concentrations only • Lack of accountability for nonuniformity
Bio-monitoring	• Objective measurements • Integrated measure of human activities • Reasonable approximation to internal dose for well-controlled studies	• May be invasive • Difficulty in being directly linked to source of pollution • Limited to measuring few components • Biological variation
Personal Monitoring	• Objective measurements • Non-invasive • Integrated measure of human activities • Samples collected in breathing zone	• Present exposure only • Limited to measuring few components • Affected by weekday activities

spouse smoked in the bedroom; the type of ventilation available during periods of exposure; whether there was exposure to ETS at workplace; and how the respondents "rate" each exposure, etc. In many studies, however, only the smoking statue of a non-smokers spouse (usually the husband) was asked and used as a surrogate for ETS exposure (Law and Hackshaw, 1996; Witschi *et al.*, 1995; Armitage *et al.*, 1997; LeVois and Layard, 1994; Coultas *et al.*, 1989).

Although the administration of questionnaires is by far the most cost-effective and convenient, the subjective nature of questionnaires renders the accuracy of the data obtained highly uncertain. In addition, several potential biases exist in the method. These include: investigator biases, misclassification of both smoking and ETS exposure status, need to rely on the memory of respondents, and the use of proxy respondents. For example, when respondents are asked to recall and quantify exposures that may or may not have occurred decades ago, memory could fail or be modified by perceptions. In instances where the subject is deceased, and a family member is asked to complete the questionnaire, incorrect information might be gathered. The effect of smoker misclassification may be especially significant in communities or ethnic groups where smoking is considered less socially acceptable. In a recent study involving married Japanese women, a significant discrepancy was reported to exist between subjective answers given on smoking status and ETS exposure and the objective analyses of urine cotinine (a marker of recent tobacco smoke exposure) in these women (Lee, 1995). It appears that the smoker misclassification rate of greater than 10% reported for Japan in this study is 2.5 to 3.5 times higher than that reported for studies involving Western populations (Riboli *et al.*, 1990; Lee and Forey, 1996). In a study on the misclassification of smoking status among Southeast Asian immigrants, Wewers *et al.* (1995) reported that almost 70% of female smokers from Cambodia misreported themselves as nonsmokers (as determined by saliva cotinine levels of more than 14 ng per ml). Such a bias will tend to elevate the RRs, e.g., for lung cancer, associated with exposure to ETS.

In summary, although questionnaires give an indication as to whether the subject was exposed to ETS in a set indoor environment, it does not and cannot give precise and objective information about the concentration and the time of people's exposure to ETS (Coultas *et al.*, 1989, 1990; Lee, 1995; Riboli *et al.*, 1990; Lee and Forey, 1996; Wewers *et al.*, 1995)

Area Monitoring. Stationary instruments have been used in numerous studies to measure the concentration of ETS constituents, such as nicotine and respirable suspended particles (RSP) in a variety of indoor settings (Guerin *et al.*, 1992). The method usually involves placing an airsampling device in a fixed location of a room. The device may have a passive, active, or continuously integrative design, allowing air samples to be taken for a period of time from minutes to days and weeks. Such measurements have been carried out in both simulated experimental situations and actual indoor environments. Collectively this type of analysis has demonstrated that concentrations of indoor air contaminants are affected by the interaction of a number of factors related to the generation, dispersal, and elimination of the contaminants. Although convenient and objective, the area monitoring method does have limitations. For example, the measured concentration of ETS constituents in a given indoor environment is not necessarily an objective measure of a person's exposure to those constituents since the on-site device is not designed to monitor: (i) the length of time a person is in the room; (ii) the person's activities whilst in the room; (iii) heterogeneity of ETS concentrations in the room due to incomplete mixing of indoor air; and (iv) improper placement of samplers.

In conclusion, although data from stationary, on-site measuring devices have provided useful information on the concentrations of indoor air contaminants, both from ETS and other sources, the information obtained cannot accurately reflect the exposure of people to ETS.

Bio-monitoring. Indicators of exposure to ETS may also be obtained by measuring the concentration of ETS constituents or metabolites, such as nicotine, cotinine, or macromolecular adducts in a person's blood, saliva or urine (Hammong *et al.*,

1987; Benowitz, 1996; Maclure *et al.*, 1989). Biomonitoring can, in principle, overcome problems that stem from an individuals incorrect recall or lack of awareness of exposure, as well as from variations in individual uptake or metabolism of the exposed agent(s). It should be emphasized, however, that the relationship between a given biomarker and exposure is complex and may vary according to the individual's dietary habits and other lifestyle factors. Moreover for diseases with multiple or unknown causes like lung cancer and cardiovascular diseases, the identification of an appropriate biomarker for ETS exposure itself represents a significant challenge.

Nicotine and its metabolite cotinine, both considered to be tobacco-specific by many, are two of the most widely used biomarkers for active smoking and short-term ETS exposure. However, measuring nicotine and cotinine for estimating a person's exposure to ETS poses several limitations. First, both nicotine and cotinine have relatively short half-lives and therefore can only be used for estimating exposure during the last few hours or days prior to sample collection. Also, nicotine in ETS mainly exists in the vapor phase and has different dispersion characteristics from ETS particles (e.g., a tendency to adhere to indoor surfaces), while nicotine is a particulate phase constituent in mainstream smoke. As a result ETS exposure as estimated according to the nicotine/ cotinine concentrations in the body fluids may not accurately reflect the exposure to ETS particles. Second, the assumption that nicotine is tobacco- specific has been questioned (Idle, 1990; Sheen, 1988; Davies *et al.*, 1991), since plant sources other than tobacco, e.g., members of the Solanaceae family such as eggplant, tomato, and green pepper, which are common dietary components, are known to contain nicotine. Tea has also been identified as a source of dietary nicotine (Sheen, 1988). Davies *et al.* (1991) measured nicotine in a number of teas and foods, and reported nicotine levels of up to 285 ng/g wet weight. Third, cotinine is only one of the metabolites of nicotine and it has been reported to show considerable individual variability in controlled nicotine exposure studies (Idle, 1990). Fourth, concentrations of biomarkers may correlate poorly to levels of ETS exposure since the measured levels may be affected by individual metabolic and clearance patterns, age, breathing pattern, previous exposure to other materials, etc. These and other limitations of nicotine/cotinine as markers for ETS exposure illustrate the importance of careful interpretation of biomarker data in estimating exposure.

Personal Monitoring. The use of personal monitors is a relatively new application of a well-known and accepted technology. Personal monitors have long been used in occupational settings to determine workers exposure to substances, especially those for which maximum permissible workplace exposure limits exist. Personal monitors can be as simple as a badge fixed to the clothing of a person or as complex as a multiport filter holder that is coupled to a battery-powered active pump fixed to the belt or the back of the person whose air is being monitored. The simple badge monitors are useful for monitoring single components in the air but are not well suited for investigating complex mixtures such as ETS. In addition, these monitors depend for their accuracy on the air movement naturally around the monitored person. Vast differences in the airflow around the monitored person can substantially distort the result from this type of monitor.

Recent Studies on Exposure to ETS Using Personal Monitoring Sampling Devices

To circumvent these problems, personal air samplers or monitors with a multiport filter and collector tube that can trap several constituents in the air were developed (Ogden *et al.*, 1996) and used in personal monitoring studies in different cities around the world (Jenkins *et al.* 1997; Phillips *et al.*, 1994; Phillips *et al.*, 1997; Phillips *et al.*, 1996a; Phillips *et al.*, 1996b; Phillips *et al.*, 1996c; Phillips, 1997). Such samplers are attached to a pump that ensures consistent airflow into the device. The air-sampling inlet of the monitor is situated in the person's breathing zone while he/she engages in his/her normal activities in his/her normal environment (Jenkins *et al.* 1997; Phillips *et al.*, 1994; Phillips *et al.*, 1997; Phillips *et al.*, 1996; Phillips *et al.*, 1996). The personal monitor

accompanies the person to the sitting room, the bedroom, the kitchen, the car, the elevator, the workplace, the park and all the other settings in which he/she spends time. As a result, the sample collected during a specified time is representative of that person's actual exposure.

As of July 1997, personal monitoring studies have been reported to be completed in three Asian cities (Beijing, Hong Kong, Kuala Lumpur) (Phillips, 1997), nine European cities (Barcelona, Basle, Bremen, Harrogate, Lisbon, Paris, Prague, Stockholm, and Turin) (phillips *et al.*, 1994; 1996a; 1996b; 1997) Sydney, (Phillips *et al.*, 1996c) and sixteen US cities geographically distributed around the United States (Jenkins *et al.*, 1997). In each of these studies at least 150 volunteers wore personal monitors over a 24-hour period. From the collected samples, levels of exposure for nicotine, respirable suspended particles (RSP), ETS particles (calculated as solanesol [SolPM], fluorescence [FPM] and ultra-violet [UVPM] particular matter), were determined. In some studies the levels of various volatile organic compounds (VOCs) were also measured (Phillips *et al.*, 1996b; Phillips *et al.*, 1996c). In each of the reported studies subjective evaluation of exposure to ETS was also assessed by questionnaire and the current smoking status of the subjects was verified by measuring their saliva cotinine levels both before and after the monitoring session. The same research group from Covance (formerly Corning Hazelton) carried out all the studies outside the United States (phillips *et al.*, 1994; 1996a; 1996b; 1996c; 1997; phillips, 1997) to ensure the results are comparable. A similar study design was also used for the US research team (Jenkins *et al.*, 1997). The major findings that were reported to date for four of the studies in peer reviewed publications (Jenkins et al., 1997; phillips *et al.*, 1994; 1996a; 1997) are summarized below. It should be pointed out that the exposures referred to as "in smoking homes" and "in nonsmoking homes" below include all away-from-work exposures (although mostly in homes) for subjects who are living in smoking and nonsmoking homes, respectively.

ETS exposure in Stockholm. Both the levels of RSP and ETS particles in Stockholm, calculated as SolPM (Phillips et al., 1996a), were reportedly among the lowest of all the cities participating in the personal monitoring studies. The cities include 4 additional European cities, Hong Kong, Beijing, Kuala Lumpur, Sydney (Phillips *et al.*, 1994; 1996b; 1996c; Phillips, 1997) and sixteen US cities (Jenkins *et al.*, 1997). For nonsmokers in Stockholm, who are not working, the median concentrations of ETS particles were reported to be 17$\mu g/m^3$ in smoking homes and below the limit of detection of 0.23$\mu g/m^3$ in non-smoking homes. For working nonsmokers, however, the median levels of ETS particles were reported to be 1.4 and 1.1$\mu g/m^3$ in smoking homes and workplaces, respectively, and below the limit of detection of 0.37$\mu g/m^3$ and 0.77 $\mu g/m^3$, respectively, in non-smoking homes and workplaces. The appreciable difference in the reported median exposure of ETS particles in smoking homes between nonworking and working nonsmokers could reflect the different number of hours spent at homes and away-from-work by these two groups of individuals. The median RSP levels in smoking and non-smoking homes were reported to be 24 and 19$\mu g/m^3$, respectively, while those in smoking and non-smoking workplaces were both reported to be 16$\mu g/m^3$. Thus, ETS particles account for 5.8% of the total RSP in smoking homes for working subjects.

For non-working subjects the median nicotine levels were reported to be 1.1$\mu g/m^3$ in smoking homes, and below the limit of detection of 0.09$\mu g/m^3$ in non-smoking homes. For working subjects the median nicotine levels were reported to be 0.15 $\mu g/m^3$ and below the limit of detection of 0.14$\mu g/m^3$ in smoking and non-smoking homes, respectively. The median nicotine levels in both smoking and non-smoking workplaces were reported to be below the limit of detection of 0.29$\mu g/m^3$).

Personal monitoring study in sixteen US cities. In the sixteen US cities (Jenkins *et al.*, 1997), the median levels of ETS particles, calculated as SolPM, were reported to be 1.25$\mu g/m^3$ in smoking homes and below the limit of detection of 0.14$\mu g/m^3$ in nonsmoking homes. The median levels of ETS particles in both smoking and nonsmoking workplaces were reported to be below the limit of detection. The median RSP levels were reported to be 27$\mu g/m^3$ in smoking homes and 16$\mu g/m^3$ in non-

smoking homes. The median RSP levels in smoking and nonsmoking workplaces were reported to be 25 and 13$\mu g/m^3$, respectively. In smoking homes ETS particles account for 4.6% of the total RSP. For individuals in the most exposed group, i.e., those who live and work in smoking environments, the average concentration of ETS particle (3.76$\mu g/m^3$) accounts for 11.9% of the average RSP concentration (33.6$\mu g/m^3$).

The median concentrations of nicotine were reported to be 0.79$\mu g/m^3$ in smoking homes and 0.03 $\mu g/m^3$ in nonsmoking homes. The median concentrations of nicotine were reported to be 0.28 and 0.04$\mu g/m^3$ in smoking and nonsmoking workplaces, respectively.

The data reported for this large study of US cities are similar to the results reported for Stockholm, with among the lowest ETS and RSP concentrations ever reported for cities with published personal monitoring study results. It is worth noting that the median nicotine levels in the workplace reported in this study is only 30-60% of the estimates given by the US Occupational Safety and Health Administration (OSHA) and 15-20% of OSHAs estimates for the most heavily exposed workers.

ETS Exposure in Barcelona. For nonsmokers in Barcelona (Phillips *et al.*, 1997), who are not working, the median concentrations of ETS particles, calculated as SolPM, were reported to be 11$\mu g/m^3$ in smoking homes and 1.0$\mu g/m^3$ in non-smoking homes. For working nonsmokers the median concentrations of ETS particles were reported to be 21 and 37$\mu g/m^3$, respectively, in smoking homes and workplaces, and 2.2$\mu g/m^3$ and 2.6$\mu g/m^3$ in nonsmoking homes and workplaces, respectively. The median RSP concentrations in smoking and nonsmoking homes were reported to be 85 and 40$\mu g/m^3$, respectively, while those in smoking and non-smoking workplaces were reported to be 94 and 52$\mu g/m^3$, respectively. ETS particles accounted for 25% and 13% of the total RSP concentrations in smoking homes for working and non-working individuals, respectively.

For non-working subjects the median nicotine concentrations were reported to be 0.74$\mu g/m^3$ in smoking homes and 0.11$\mu g/m^3$ in non-smoking homes. For working subjects the median nicotine concentrations were reported to be 0.86$\mu g/m^3$ and 0.17$\mu g/m^3$ in smoking and non-smoking homes, respectively. The median nicotine concentrations in smoking and non-smoking workplaces were reported to be 2.4 and 0.71$\mu g/m^3$, respectively.

ETS exposure patterns in Barcelona differ from those reported for Stockholm and the sixteen US cities; the highest median ETS and RSP levels were found in smoking workplaces, whereas in both the Stockholm and the US cities, smoking homes reportedly had the highest ETS and RSP levels.

Summary of the personal monitoring studies. Personal monitoring studies have been conducted in 29 cities from four continents that differ significantly with respect to lifestyle, living standards, degree of reliance on agricultural and industrial-based economies, and the type and severity of indoor/outdoor pollution. Results from the published personal monitoring studies show that there are differences in the ETS exposure patterns among subjects in different cities but the actual overall levels of exposure to ETS are low. For cities in which personal monitoring study results were published, nonsmokers in Barcelona, both living and working in smoking environments, were reported to have the highest levels of exposure to ETS. These individuals would be expected to be exposed to less than 12 cigarette equivalents (CE) per year based on the median levels of either ETS particles or nicotine, and assuming an average breathing rate of 0.85m^3 per hour (Phillips *et al.*, 1996c). The estimated CE/year amounts to less than 0.2% of the cigarettes an active smoker, with an average daily consumption of one pack, would consume in a year. Most of the nonsmokers in Barcelona and all other cities for which personal monitoring results are available would be exposed to even lower levels of ETS. It should be noted that the expression of a person's exposure to ETS as CE assumes that the subjects were exposed to these median levels throughout the year.

There are large differences among cities in the percentage of current smokers who misreport themselves as nonsmokers, as judged by a salivary cotinine cut-off concentration of 15 ng/ml (for US cities) and 25 ng/ml (for all other cities). The re-

ported misclassification was ca. 4% in Stockholm (Phillips *et al.*, 1996a) and US cities (Jenkins *et al.*, 1997), between 11-18% in Barcelona (Phillips *et al.*, 1997), and 13% in Harrogate (Phillips *et al.*, 1994).

These studies also showed that although a good correlation exists between premonitoring saliva cotinine levels and SolPM values, the correlation was poor when postmonitoring cotinine and SolPM were compared. Thus, single point saliva cotinine measurements alone should not be considered as reliable markers for ETS exposure, particularly when the levels of exposure are low. Since the personal monitoring study results show that ETS particles make a minor contribution to indoor RSP, the higher levels of RSP observed in smoking environments cannot be solely explained by contribution from ETS.

Conclusion

Personal monitors clearly represent a significant technological advance and improvement that allow objective measurements of a person's actual exposure to ETS. Being able to account for both the concentrations of several ETS constituents and the time that a person is exposed to ETS, personal monitoring is currently the most reliable method for measuring a person's actual exposure to ETS. It should be pointed out, however, that these measurements still represent short-term exposures to ETS. Further studies are needed to validate whether they are realistic approximations of long-term exposures. The potential differences in people's activity patterns during workdays vs. weekends may affect their ETS exposure levels. Such a possibility needs to be investigated in future personal monitoring studies.

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References

Armitage, A.K., J.R. Ashford, J.W. Gorrod and F.M. Sullivan (1997) Environmental tobacco smoke - is it really a carcinogen? *Med. Sci. Res.* 25:3-7

Aviado, D.M. (1996) Cardiovascular disease and occupational exposure to environmental tobacco smoke. *Am. Ind. Hyg. Assoc. J.* 57: 285-294.

Benowitz, N.L. (1996) Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol. Rev.* 18: 188-204.

Coultas, D.B., G.T. Peake and J.M Samet (1989) Questionnaire assessment of lifetime and recent exposure to ETS. *Am. J. Epidemiol.* 130: 338-347.

Coultas, D.B., J.M. Samet, J.F. McCarthy and J.D. Spengler (1990) A personal monitoring study to assess workplace exposure to environmental tobacco smoke. *Am. J. Public Health* 80:988-990.

Davies, R.F., M.F. Stiles, J.D. DeBethizy and J.H. Reynolds (1991) Dietary nicotine: a source of urinary cotinine. *Food Chem. Toxicol.* 29821-827.

Glantz, S.A. and W.W Parmley (1991) Passive smoking and heart disease. Epidemiology, physiology, and biochemistry. *Circulation* 83: 1-12.

Guerin, M.R., R.A. Jenkins and B.A. Tomkins (1992) The chemistry of environmental tobacco smoke: composition and measurement. *Lewis Publisher, Inc.*

Hammong, S.K., B.P. Leaderer, A.C. Roche and M. Schenker (1987) Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmos. Environ.* 21: 457-462.

Idle, J.R. (1990) Titrating exposure to tobacco smoke using cotinine-a minefield of misunderstandings. *J. Clin. Epidemiol.* 43: 313-317.

Jenkins, R.A., A. Palausky, R.W. Counts, C.K. Bayne, A.B. Dindal and M.R. Guerin (1997) Exposure to environmental tobacco smoke in sixteen cities in the United States as determined by personal breathing zone air sampling. *J. Exposure Analysis and Environ. Epidemiol.* 6:473-502.

Law, M.R. and A.K. Hackshaw (1996) Environmental tobacco smoke. *Br. Med. Bull.* 52: 22-34.

Lee, PN. (1995) "Marriage to a smoker" may not be a valid marker of exposure in studies relating to environmental tobacco smoke to risk of lung

cancer in Japanese non-smoking women. *Int. Arch. Occup. Environ. Health* 67: 287-294.

Lee, P.N. and B.A. Forey (1996) Misclassification of smoking habits as a source of bias in the study of environmental tobacco smoke and lung cancer. *Statistics in Medicine* 15: 581-605.

LeVois, M.E. and M.W Layard (1994) Inconsistency between workplace and spousal studies of environmental tobacco smoke and lung cancer. *Regul. Toxicol. Pharmacol.* 19: 309-316.

Maclure, M., R.B. Katz, M.S. Bryant, P.L. Skipper, and S.R. Tannenbaum (1989) Elevated blood levels of carcinogens in passive smokers. *Am. J. Public Health* 79: 1381-1384.

Ogden, M.W., D.L. Heavner, T.L. Foster, K.C. Maiolo, S.L. Cash, J.D. Richardson, P. Martin, P.S. Simmons, F.W. Conrad and P.R. Nelson (1996) Personal monitoring system for measuring environmental tobacco smoke exposure. *Environ. Technology* 17: 239-250.

Phillips, K., D.A. Howard, D. Brown and M. Lewsley (1994) Assessment of personal exposures to environmental tobacco smoke in British nonsmokers. *Environment Intl.* 20: 693- 712.

Phillips, K., M.C. Bentley, D.A. Howard and G. Alvan (1996a) Assessment of air quality in Stockholm by personal monitoring of nonsmokers for respirable suspended particles and environmental tobacco smoke. *Scand. J. Work. Environ. Health* 22 (Suppl 1): 3-24.

Phillips, K., M.C. Bentley, D.A. Howard, G. Alvan and A. Huiei (1997) Assessment of air quality in Barcelona by personal monitoring of nonsmokers for respirable suspended particles and environmental tobacco smoke. *Environ. Intl.* 22: 173-196.

Phillips, K., D. Howard, M. Bentley and G. Alvan (1996b) Assessment of air quality in Europe by personal monitoring of nonsmokers in homes/workplaces for respirable suspended particles(RSP), environmental tobacco smoke (ETS) and volatile organic compounds (VOCs) inside and outside homes. *Proceedings Indoor Air '96. The 7th International Conference on Indoor Air Quality and Climate.* Nagoya, Japan, Volume 1: 495-500.

Phillips, K., M. Bentley, D. Howard, J. Cook and G. Alvan (1996c) Air quality in Europe, China, South East Asia and Australia. Presentation, 2nd International Conference on Environmental and Industrial Toxiciology, December 12, 1996, Bangkok, Thailand.

Phillips, K. (1997) Monitoring personal exposures to environmental air pollutants. Presentation, International workshop on risk assessment and good epidemiological practices. July 14-17, Guangzhou, China.

Riboli, E., S. Preston-artin, R. Saracci, N.J. Haley *et al.* (1990) Exposure of nonsmoking women to environmental tobacco smoke: a 10-country collaborative study. *Cancer Causes Control* 1:243-252.

Reynolds, P. and E.T. Fontham (1995) Passive smoking and lung cancer. *Ann. Med.* 27:633-640.

Rosenbaum, W.L., T.D. Sterling and J.J. Weinkam (1996) A critical examination of OSHA's assessment of risk associated with workplace exposure to environmental tobacco smoke. *Regul. Toxicol. Pharmacol.* 23: 233-240.

Sheen, S.J. (1988) Detection of nicotine in foods and plant materials. *J. Food Sci.* 53: 1572-1573.

Wewers, M.E., R.K. Dhatt, M.L. Moeschberger, R.M. Guthrie, P. Kunn and M.S. Chen (1995) Misclassification of smoking status among Southeast Asian adult immigrants. *Am. J. Respir. Crit. Care Med.*, 152:1917-1921.

Witschi, H, K.E. Pinkerton, C.R. Coggins, A. Penn and G.B. Gori (1995) Environmental tobacco smoke: experimental facts and societal issues. *Fundam. Appl. Toxicol.* 24: 3-12.