Passive Smoking in Children
The Importance of Parents’ Smoking and Use of Protective Measures

AnnaKarin Johansson
To Göran, Stefan, Ulf and Daniel
My aim was to contribute to the future and not to blame the past
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Abstract
Passive smoking has been recognised as a health hazard, and children are especially vulnerable. The general aim of this thesis was to describe and analyse the importance of parents’ smoking and smoking behaviour for children’s tobacco smoke exposure. The studies were conducted in the South-East part of Sweden and pre-school children and their parents constituted the study samples. Five studies are described in six papers. Smoking prevalence among parents (14%) and commonly used measures of protection were surveyed. An instrument designed to measure children’s tobacco smoke exposure in the home was developed and validated. It was used on 687 families with a smoking parent and a child 2½-3 years old, included in a prospective cohort study on environmental variables of importance for immun-mediated diseases ABIS (All Babies in South-East Sweden). Almost 60% of the parents stated that they always smoked outdoors with the door closed, 14% mixed this with smoking near the kitchen fan, 12% near an open door, 7% mixed all these behaviours and 8% smoked indoors without precautions. The smoking behaviours were related to the children’s creatinine adjusted urine cotinine. All groups had significantly higher values than had children from non-smoking homes, controls. Outdoor smoking with the door closed seemed to be the best, though not a total, measure for tobacco smoke protection in the home.

Most parents were aware of the importance of protecting children from tobacco smoke exposure but all were not convinced of the increased risk for disease for exposed children. The majority of parents were not satisfied with the smoking prevention in health-care and 50% did not think that their smoking was of any concern to the child health care nurse.

Further research is warranted to describe if the difference in exposure score related to smoking behaviours is related to different prevalence of disease. Efforts are needed to convince those who still smoke indoors that tobacco smoke exposure influence children’s health and that consequent outdoor smoking with the door closed seemed to give the best protection.

Key words: ETS, infant, child, cotinine, smoking behaviour, protective measures, parents, home, tobacco, child health care, ABIS
ABBREVIATIONS

ABIS  All Babies in South-East Sweden
BC    Before Christ
CCR   Cotinine/Creatinine Ratio
CHC   Child Health Clinic
CHD   Cardiovascular Heart Disease
CI    Confidence Interval
CO    Carbon monoxide
ETS   Environmental Tobacco Smoke
EU    European Union
LLQ   Lowest Level of Quantification
OR    Odds Ratio
RSP   Respirable Suspended Particles
WHO   World Health Organisation

Definitions:
Dependent children  individuals 0-19 years old

Immigrants  individuals not born in Sweden

Indoor smoker  a smoker, smoking sometimes or always anywhere indoors, including standing near an open door or window or near the kitchen fan (I,II,VI)

Indoor smoker  a smoker, smoking anywhere indoors, at dinner table or near the TVset (III–V).

Outdoor smoker  a smoker always smoking outdoors with the door closed

Passive smoking  the inhaling of ETS; diluted sidestream smoke and exhaled mainstream smoke

Pre-school children  individuals 0-6 years old

School children  individuals 7-19 years old

Smoker  daily and occasional smokers

Smoking behaviour  active choice of places when smoking
LIST OF ORIGINAL PAPERS

This thesis is based on the following papers, which will be referred to in the text by their roman numerals.


II  **Does having children affect adult smoking and behaviours at home?** Johansson AK, Halling A, LinQuest study group. Tobacco Induced Diseases 2003; 1: 175–83, copyright (2003), with permission from PTID society


IV  **When does exposure of children to tobacco smoke become child abuse?** Johansson AK, Hermansson G, Ludvigsson J. The Lancet 2003; 361: 1828 [letter], copyright (2003), with permission from Elsevier

V  **How should parents protect their children from ETS exposure in the home?** Johansson AK, Hermansson G, Ludvigsson J. Accepted for publication in Pediatrics.

VI  **Attitudes to children’s tobacco smoke exposure among smoking and non-smoking parents and their opinions on how the issue is handled in health care.** Johansson AK, Hermansson G, Ludvigsson J. Revised and resubmitted to J Pediatric Health Care
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INTRODUCTION

Smoking is a well-known world-wide public health problem (1). The adverse health effects from tobacco smoking were confirmed in the 1950’s (2). Around 5 000 000 premature deaths per year is estimated to be caused by tobacco smoking (3). Since the 1980’s the adverse health effects from tobacco smoke on non-smokers spending time in environments polluted by tobacco smoke, have been known. Passive smoking is now regarded as the third health threat in the world, after smoking and alcohol abuse. Though the individual risks for disease are moderately increased, the exposure to tobacco smoke has a major health impact since almost half of the children in the world are exposed (1).

There have been a plethora of interventions defeating tobacco use which have been to some extent successful: Smoking prevalence has decreased and the legislation restricting smoking and tobacco purchase has become of considerable proportions in the Western world (4). However, still new young smokers are recruited daily. Globally, both tobacco consumption and tobacco production increase (5).

Children are especially vulnerable to tobacco smoke exposure. This starts during pregnancy and breast-feeding in infancy when they share their mothers active or passive smoking and is followed by passive smoking during childhood. Children’s body and organs are developing and growing and thus more sensitive to adverse influence and children have higher relative ventilation rates leading to higher internal exposure than adults have. Further young children have no possibility to escape from an ETS (Environmental Tobacco Smoke) polluted environment by their own will. Due to new legislation restricting smoking in public arenas and changed social norms for tobacco smoking, children’s tobacco smoke exposure almost exclusively takes place in their homes (6).

My intention with these studies has been to further increase the knowledge on which measures of protection, that smokers, who do not want to, or are not able to stop smoking, should take to prevent children’s tobacco smoke exposure. To my experience most parents, independently of if they have chosen to continue to smoke or not, are very anxious to protect their children. Therefore parents take different measures to protect their children. Some of the methods are awkward and require sacrifices by the parents. Still the knowledge on how effective these measures are has been scarce.
The comprehensive aim of this thesis has been to increase the understanding of smoking and smoking behaviour among parents of young children and to assess the effectiveness of measures taken to protect the children from tobacco smoke exposure in the home.
BACKGROUND

THE HISTORY OF SMOKING

The history of smoking starts among the Native Americans who used it for ceremonial purposes 5000 years BC. Christopher Columbus first brought tobacco to Europe from the West Indies in 1492. From the beginning it was used for medical purposes and in history it is mentioned when the queen of France, Catherine of Medici, was cured from stomach pains by tobacco. She got the tobacco from Jean Nicot and named it “Nicotiana”. Soldiers during the big European wars spread the use of tobacco, mostly used as snuff or smoked in pipes. It was not until the Crimean War that cigarettes became more common. When the first cigarette machine was constructed in 1870 cigarette smoking flourished. This was also the start for the big tobacco companies (7).

Cigarette smoking was from the beginning a masculine habit and spread among soldiers during World War I and II. Women began to take up smoking during and after World War II, thus putting children in closer contact to ETS. The era of the well-educated and career-oriented women began; smoking became a sign of independence and is seen as part of the women’s liberation. Cigarette smoking then spread to other groups in society and has now become most prevalent among under privileged women with a short education (8). World Health Organisation (WHO) reports that Sweden (19%) besides Norway (29%) and New Zealand (25%) are the exclusive countries in the world where women smoke to the same extent as men (5).

Smoking cessation has roughly followed the same path as the introduction of the habit in society. It was the well-educated and prosperous men that started the trend; working class men and then the well-educated women followed them. At the turn of the century smoking, at least habitual smoking became a sign of lower class and lack of character. It was considered as the largest separate health risk in Sweden and as a state of illness with a ICD-10 (International Classification of Diseases) number (8, 9). Nicotine addiction is classified in the group (F17) for mental and behavioural disorder depending on the use of a psychoactive substance (10).

During the last decade smoking prevalence has decreased in the Western world. However, this is compensated by an increase in the developing countries. Also, in the developing countries men started smoking first and women followed soon thereafter. Globally the tobacco consumption as well as the production is growing and the women in Asia and Africa are now the main target group for
the tobacco companies (5). Though smoking prevalence in the Western world decreases, smoking has kept an aura of tough and smart glamour, and 80-100 000 new young smokers are recruited daily, according to WHO (Gro Harlem Brundland 31-05-2000). Totally about 1/3 of the adult population smokes and WHO has calculated that 1000 cigarettes are manufactured per year per person, including women and children (5).

Society has tried to influence smoking habits in different ways during the years. Smoking has been looked upon with great indignation and was regarded as immoral in the early 20th century. Later smoking became highly accepted, and in social life many rules on how to offer cigarettes and perform smoking were included. After the reports on adverse health effects from smoking the anti-smoking debate was intensified in the 1960’s and was accelerated in the 80’s when it was shown that also passive smoking was a health hazard. During the 90’s numerous conventions, national as well as international, have dealt with the smoking issue. National as well as international authorities, like WHO and EU (European Union), have made up rules and recommendations for how the "pandemic of smoking" can be defeated. Many countries have passed laws on smoke free arenas, rules for cigarette commerce and public health interventions to control tobacco use. These legal proceedings as well as other measures have, to some extent, been successful (8). Parts of USA and Australia, and Sweden have done well and have the lowest prevalence of smokers (developing countries excluded) in the beginning of the 21st century (Australia 19.5%, Sweden 19%), (5) and California 18% in 1997 (11). In these areas smoking has become the habit of the short educated and unprivileged parts of the population.

In Sweden the passing of the Tobacco Law (12) can be seen as a milestone. After having been analysed for a decade the first version was passed in 1993 and in 1994, 1997 and 2002 it was tightened up. The law regulates where smoking is prohibited, the printing of warning labels on cigarette packets, and the advertising and cigarette purchase by juveniles (<18 years of age). In EU big efforts are made in trying to standardise legislation and aims for tobacco prevention (4).

Numerous anti-smoking interventions have been made, mostly during the 90’s, from both public and private initiatives. They have focused on help for smoking cessation or on preventing young people from starting to smoke. There have also been campaigns focused on the importance of protecting the environment and especially children from tobacco smoke exposure (4). One example is “Smoke-free children”, a long-term, nationwide project starting in 1992 (13). It has included seminars, information material, and a comprehensive education of
midwives and nurses in CHC (Child Health Care). The aim of the courses was to increase the awareness of tobacco issues and the ability to discuss smoking with the parents using a client-centred approach. The parents’ ability to arrange a tobacco-free environment for children by enhancing their self-efficacy was focused (14). The nurses got a manual (15) to use in these meetings with smoking parents. The manual contained questions suggested to start with: 1) what do you know about passive smoking, 2) suggest to the parents that they register the smoking in the home and how close to the child smoking is performed, 3) discuss the survey with the parents and ask them for possible improvements, 4) support every improvement, 5) be especially aware of women who have stopped smoking during pregnancy and are greatly at risk to start smoking again. The project has also included the development of national statistics on smoking among pregnant women and parents of pre-school children. The statistics are based on the documentation of parents’ smoking in the health record of each child, made by the CHC nurses (figure 1). This has been done since 1996.
Surveys made by the National Board of Welfare both in 1997 and 1999 (16, 17) indicated that the awareness of the tobacco issue had increased and the tobacco prevention work had been intensified during the 90’s – the period when “Smoke-free children” was disseminated. It has been difficult to make a proper evaluation of the effect of the interviewing method since it has been so well spread over the country. However, the method has been described and evaluated in Arborelius & Bremberg (14) and Fossum, Arborelius and Bremberg (18) and was shown to have a positive effect on parental smoking behaviour.

**SMOKING CAUSES ADVERSE HEALTH EFFECTS**

The understanding of the health risks associated with smoking was established in the 60’s. Health risks had, however, been suspected earlier. In the 30’s the idea of an association between lung cancer and smoking had been risen, and with Doll & Hill (2) the connection was demonstrated. His study has been followed by a large quantity of studies from all over the world. Not only lung cancer, but most other forms of cancer, lung disease and cardiovascular diseases, osteoporosis, gastric ulcer, infertility and goitre have been recognised as being caused or associated to smoking (1).
The number of premature deaths caused by smoking was calculated in Lancet (3). In the year 2000, 5 million individuals in the world died due to tobacco use, 2.43 million in the industrialised, and 2.41 million in the developing countries. These figures are expected to rise since the global prevalence of smokers is on the increase. WHO estimated that there are about 1 billion smokers in the world; one third of all people more than 15 years old. Thirty percent of all deaths among men 35–69 years old in developed countries are estimated to be caused by smoking. Specifically, smoking causes: 90-95% of all lung cancer deaths, 75% of chronic lung disease deaths, 40-50% of all cancer deaths, 35% of cardiovascular disease deaths and more than 20% of vascular disease deaths (1). In Sweden the number of deaths related to smoking among the 35-84 year age group was reported to be 6412, in 1999-2000 (5).

**PASSIVE SMOKING**

Passive smoking is defined as the non-smokers inhalation of tobacco smoke produced by the active smoking of others (19). In this thesis it is equivalent to “being exposed to second-hand smoke or tobacco smoke or ETS“ (Environmental Tobacco Smoke).

ETS is composed of the diluted tobacco smoke from the burning ends of cigarettes, pipes and cigars (sidestream smoke) and the exhaled smoke from smokers (mainstream smoke). It is a complex mixture of gas and particle-phase chemicals, and the composition changes during its dilution and distribution in the environment and upon ageing. The sidestream smoke is shown to contain about the same hazardous substances as mainstream smoke. Quantitatively, however, side stream smoke contains much more of the different chemical constituents, varying from double to the hundredfolded amounts for the different chemical constituents. This is due to the lower burning temperature between the “puffs”. More than 75% of the nicotine emitted from a cigarette is emitted into the air as sidestream smoke (20).

Variables shown to be of importance for the intensity of passive smoking are number of habitual smokers/100 m$^3$ ($D_{hs}$) and air exchange rate in air changes per hour ($C_v$). Repace et.al. (21) made a mathematical model of this connection: $N=22 \frac{D_{hs}}{C_v}$, where $N$ is the equilibrium nicotine concentration in µg/m$^3$. Repace et. al. (21) have also shown that it is impossible to protect non-smokers by ventilation. They calculated that tornado-like levels (50 000 litres per second per occupant) of airflow were needed to achieve the “de minimis risk level” (the level of maximum acceptable risk, occupational regulatory levels) for heart disease and lung cancer among non-smokers working in an office.
Young children’s ETS exposure mostly takes place in their homes and the main source is parental smoking (22, 23).

**PASSIVE SMOKING CAUSES ADVERSE HEALTH EFFECTS**

The effects of maternal smoking during pregnancy were well documented by the mid-1960s with a number of studies showing reduced birth weight for children born to smoking mothers (24). Later it was also associated to prematurity (25), sudden infant death syndrome (26), and reduced lung capacity (27).

In the beginning of the eighties further studies on the adverse health effects of ETS exposure were reported. One of the firsts was Hirayama (28) who found that non-smoking wives of heavy smokers had a significantly elevated risk of lung cancer. In 1986 two important reports, Surgeon General (29) and IARC (International Agency for Research on Cancer) (30), concerning the connection between passive smoking and adverse health effects, were published. They have been followed by numerous studies on the subject and passive smoking has been considered to be number three of preventable causes of illness and untimely death, after active smoking and alcohol abuse (1, 19). To be exposed a few times a week over the years was enough to increase the risk for CHD according to Panagiotakos (31).

Passive smoking has been shown to cause lung cancer among non-smokers (32). Studies on other cancer sites have been conflicting and no causal relationship has been established. Studies on experimental animals have, however, shown sufficient evidence for carcinogenicity of sidestream smoke condensates. IARC’s (32) conclusion from their overall evaluation that exposure to ETS is carcinogenic to humans, group 1. ETS exposure has also been causally associated to coronary heart disease (33, 34) and chronic respiratory symptoms (19). A dose-response association between cardiovascular disease and exposure level has been shown both with using frequency of exposure (35) and objective biomarkers (cotinine) (36).

Finally the adverse effects from ETS exposure were established in the Fifty-sixth World Health Assembly: WHO Framework Convention on Tobacco Control 21 May 2003 Article 8: “Parties recognise that scientific evidence has unequivocally established that exposure to tobacco smoke causes death, disease and disability” (37).

An increased likelihood of taking up smoking in adolescence, if ETS exposed as a child, has been shown (38, 39, 40). This will increase the adverse health effects from earlier passive smoking.
THE CHILD’S INCREASED SENSITIVITY TO ETS EXPOSURE

1. Children have been reported to be at higher risk than adults when exposed to ETS. Reasons suggested to be the cause of this increased vulnerability compared to adults are:

2. For the unborn child of a smoking mother or a non-smoking mother exposed to ETS, oxygen delivery may be compromised by carbon monoxide (CO) in cigarette smoke. CO binds to haemoglobin and thus reduces its oxygen carrying capacity which gives the growing foetus impaired conditions. This has been shown to cause an increased risk for low birth weight and preterm delivery (24). Smoking during pregnancy has been shown to explain about 10% of the variability in birth weight (41).

3. During the first years of a child’s life the lung development is finished as formation of the alveoli is completed and lung function increases in parallel to the increase in height. ETS exposure during this process may have lasting effects and comprise the lungs reserve capacity (27).

4. The immune system is sensitive to the influence of environmental factors, of which ETS might be one.

5. Compared with adults children have higher relative ventilation rates leading to a higher internal exposure, as measured by urinary cotinine, for the same level of external exposure (42).

6. Small children are unable to complain and unable to remove themselves from exposure. They are thus dependent on other’s measures for protection.

ADVERSE HEALTH EFFECTS FROM CHILDREN’S ETS EXPOSURE

Children’s ETS exposure has been identified as a cause of media otitis, lower respiratory tract illness, shown to worsen asthma symptoms and to have an adverse, probably irreversible, effect on lung function (43, 44, 45). Mothers’ smoking is shown to cause small reductions in children’s lung function as well as reductions in birth weight (24). Maternal smoking is also a major cause of SIDS (Sudden Infant Death Syndrome) (26). Further ETS has been shown to be a cause of chronic respiratory symptoms in school children (27). Other symptoms, shown to be associated to ETS exposure, are an increased risk for wheezing, rhinitis and infantile colic (46), a lower plasma oxidant status (47),
and an accelerated formation of arteriosclerotic plaque (32). ETS exposure has also been associated to changes in child neurodevelopment and behaviour, e.g. learning difficulties and language impairment (48). Further, though it is difficult to measure, it is also possible that exposure to ETS as a child may increase the risk for adverse health effects in adulthood, e.g. lung impairments (27), and cardiovascular disease 49).

The importance of ETS exposure on the immune system of the foetus and infant for the development of immune mediated diseases is not demonstrated. An increased risk of allergic disease and ETS exposure has been suggested (50, 51). A literature review of 36 studies did not support this (27), and other studies have shown an association between current exposure to ETS and a lowered risk for atopic disorder (52).

Postnatal ETS exposure and prenatal maternal smoking are often collinear. This fact makes it difficult to assess the true effect of postnatal exposure. To separate the effect of these two exposure forms would require a large sample of women not smoking during pregnancy and taking up smoking after delivery (48).

The judgement that an association is causal indicates that the evidence has crossed a threshold for certainty. A single study does not provide a sufficient basis for identifying a causal relationship between a risk factor and a disease. The use of the words “association” and “cause” thus demonstrate differences in how safe research results have been regarded (53).

For most of these health effects the increased individual risk is moderate. However, ETS exposure is widespread and even small increases in average individual risk result in large population risks. This can be regarded as an example of the epidemiological or prevention paradox saying that a moderate but common risk-increase for the individual causes more harm in a population than a high but less prevalent risk (54). A preventive measure which brings much benefit to the population thus offers little to each participating individual – which might result in poor motivation for the individual to take the proposed preventive measures.

There is a strong consensus among scientists and authorities that exposure to ETS causes adverse health effects. However, although the consensus is strong it has still been argued. A review article by Denson (55) claims that confounding factors like socioeconomic and diet of mother and child has not been controlled properly in studies on effects of ETS exposure.
METHODOLOGICAL CONSIDERATIONS IN RESEARCH ON SMOKING AND TOBACCO SMOKE EXPOSURE

When studying the nature of smoking in society epidemiological studies elucidating the distribution and determinants of disease frequency in human populations are used. Descriptive studies show the distribution of disease in different subgroups and the results can be used to formulate epidemiological hypotheses. In cross-sectional surveys exposure and disease have been assessed at the same point in time and the temporal relationship between variables cannot be clearly determined. Thus the presence of an association might be possible to show but a causative relationship cannot be firmly established (53).

Analytic epidemiology is used to test epidemiological hypotheses and are either observational or include an intervention. Observational studies are most often either a case-control study or a cohort study. A cohort design can provide information on the full range of health effects of a single exposure. Subjects are classified on the basis of presence or absence of exposure. Cohort studies can be either prospective or retrospective or a mix when data are collected both retrospectively and prospectively. A prospective study is most often time-consuming and expensive and the risk of loosing participants during the data sampling must be regarded but the temporal sequence between exposure and disease can be more clearly elucidated. With a retrospective design the study usually can be conducted more cheaply and quickly, but depend on relevant earlier collected data on exposure and it is most often impossible to get information on confounding factors (53).

Principal of selection of participants, big samples preferably defined after a power calculation, unambiguous definitions of conceptions, a good memory among participants and honest answers are important aspects of an epidemiological study (53).

It has been regarded difficult to demonstrate health effects caused by passive smoking. The strength of the exposure depends on several variables besides the amount of smoked tobacco, e.g. ventilation, size of room, proximity to the smoker, some of them difficult to measure and standardise. When children’s tobacco smoke exposure is to be assessed we have to rely on two primarily sources; 1) the memory and honesty of their parents and other adults in their vicinity and 2) the existing objective measurements of exposure or proper substances in the environment. Neither of these variables can be measured perfectly and their weaknesses are discussed below (56).
Questionnaires

Questionnaires have been and are the most common way of estimating ETS exposure. Numerous variants of questionnaires have been developed and used, mostly focusing on parental cigarette consumption. Different questions have been used to assess children’s ETS exposure. Some examples:

- The parents have been asked if they smoke, mother and father respectively (56).
- Parents have been asked to report number of cigarettes smoked when the child is present (57, 58, 59).
- Parents have been asked to estimate the number of hours a child has been exposed (60).
- Parents have been asked to collect cigarette butts (61).

The alternatives have their strengths and weaknesses. Jarvis (56) has outlined suggestions for items important to consider when making an instrument for assessing children’s ETS exposure. Except parents’ smoking status, cigarette consumption, smoking by child carers and visitors, day of the week, season of the year, socio-economic factors like deprivation, crowding, size of dwelling and parental education are variables that have been shown to bear an independent predictive relationship to cotinine concentrations in children.

Limitations with questionnaires are a tendency among parents to limit their children’s exposure to give socially desirable responses (62, 63) or change their “smoking behaviour” in response to measures alone (64). This risk is especially notable if the study sample consists of children with symptoms known to be associated to ETS exposure. Clark et al (65) showed a trend for the parents of asthmatic children to give more unreliable answers than parents of non-asthmatics. However, the opposite has also been reported, Callais’ study (66) indicated that parents of asthmatics tended to be more motivated to give accurate answers. There is also a risk for recall bias and a risk for misinterpreting the questions. Emerson (57) showed that the parents report on ETS exposure was more accurate for workdays compared to non-workdays, probably due to more strict routines in workdays. Brunekref et al (67) suggested that variation in parental reports might depend on instrument and population, and the age and symptom status of the children.

Objective assessment methods:
1. Airsampling

One way of measuring ETS is by air sampling. However, the complex unstable nature of ETS, which depends on several variables like ventilation, surfaces and
so on, makes it impossible to measure. The possibility is to assess some special constituents of known significance. The concentration of respirable suspended particulates (RSP) or nicotine in the air can be measured. This can be done either with personal pump-driven samplers, personal diffusion-based nicotine monitors or stationary air samplers (68, 69, 70).

RSP measures of ETS are non-specific and may include particles from fireplace, cooking and so on. They may, however, be necessary for complete estimates of disease risk, as small particles can be another source of toxic exposure (70).

2. Biomarkers
Different substances, out of the more than 4000 present in ETS, have been used to measure the strength of ETS exposure. The National Research Council in USA (71) has proposed criteria for a valid marker of ETS in the air. The marker 1) should be unique or nearly unique for ETS so that other sources are minor in comparison, 2) should be easily detectable, 3) should be emitted at similar rates for a variety of tobacco products and 4) should have a fairly constant ratio to other ETS components of interest under a range of environmental conditions encountered. Other important facts are individual differences in metabolism and excretion of the substance, if the substance is present in other sources than ETS and the sensitivity and specificity of the analytical methods used to measure the chemical (20, 72).

Some examples of used markers are presented here (20, 72):

- **CO (carbon monoxide)** has been used though it has a low specificity and sensitivity, reflect only the last hours’ exposure and emanates from many sources.
- **Thiocyanate** has also been used. It has a low sensitivity and specificity and reflect some weeks exposure. It has many dietary sources, and no difference has been shown between exposed and non-exposed.
- **Nicotine**, is a highly lipid-soluble alkaloid which has a high specificity and sensitivity. However, it has a short half-life and thus reflect only the last few hours exposure.
- Other substances like 4-aminobiphenyl-hemoglobin adduct, benzopyrene-DNA adduct, polycylic aromatic hydrocarbon-albumin adduct, urinary tobacco-specific nitrosamines have been tried but were shown to be unusable as biomarkers of ETS exposure.
- **Cotinine** is a metabolite of nicotine and has a high specificity and sensitivity and reflects the last 3-4 days’ of ETS exposure. It is the most common substance and by some researchers regarded as “the golden standard” (56). It can be measured in saliva, plasma, urine and hair.
Cotinine is metabolised from nicotine, in a two-step process involving cytochrome P450 and aldehyde oxidase. The elimination is primarily made in the hepatic metabolism and on average 70-80% of the nicotine is converted to cotinine (73). The individual variation is due to genetic variability in the activity of the conjugating enzyme (74).

Exposure to ETS causes a rapid increase in cotinine level and about 5 hours after the end of the exposure a plateau is reached. This level lasts about 12 hours, whereupon a log linear decrease is seen. According to Benowitz (20) a steady state will develop in about 5 half-lives when a drug is absorbed at a constant rate. The relatively long half-life of cotinine (~17 hours) lead to relatively constant cotinine levels throughout the day, remaining at near steady-state values. Thus an assumption of steady state for cotinine levels is reasonable when there is a daily exposure to ETS. For population studies a random cotinine measurement can be used as an indicator of daily ETS exposure (20).

According to Willers et al. (42) cotinine has been shown to have the same half-life for children as for adults and for smokers compared to non-smokers. They also found that children had a higher estimated nicotine dose and higher cotinine levels than adults.

Hovell et al (64) made a critical review of measures of ETS and discusses the lack of exactness of the available measurements, all being equally incompletely valid. It was concluded, however, that current measures, if used in combination, meet the standards necessary for larger scale epidemiological and clinical trials. They also serve well as estimates for settings that do not change often.

Analytical methods for measurement of cotinine in non-smokers are presented in table 1.
Table 1. Analytical methods for measurement of cotinine in non-smokers (20; Benowitz 1996, p 199)

<table>
<thead>
<tr>
<th>Method</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Cost</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radioimmunoassay</td>
<td>1-2 ng/ml</td>
<td>Variable</td>
<td>Low</td>
</tr>
<tr>
<td>High performance liquid chromatography</td>
<td>~1 ng/ml</td>
<td>good</td>
<td>Moderate</td>
</tr>
<tr>
<td>Gas chromatography*</td>
<td>0.1-0.2 ng/ml</td>
<td>good</td>
<td>Moderate</td>
</tr>
<tr>
<td>Gas chromatography-mass spectrometry</td>
<td>0.1-0.2 ng/ml</td>
<td>Excellent</td>
<td>High</td>
</tr>
<tr>
<td>Liquid chromatography-atmospheric pressure chemical ionization tandem mass spectrometry</td>
<td>&lt;0.05 ng/ml</td>
<td>Excellent</td>
<td>Extremely high</td>
</tr>
</tbody>
</table>

*used in the studies of this thesis

Cotinine analyses in serum has also been performed. However, when study samples include children, urine samples have been preferred. The relationship between cotinine in serum and in urine has been calculated to be approximately 1:6 (20). Further there are studies using saliva samples. Though easy to provide there are disadvantages in using saliva. Artificially, high estimates of cotinine in saliva compared with serum levels have been shown. This has been explained by the ability of the salivary glands to concentrate cotinine (75).

Cotinine can also be analysed in hair, thus reflecting months of exposure (the cm hair closest to the scalp represent the exposure in the most recent month). Al-Delaimy et al. (76) and Zahlsen & Nilsen (77) have demonstrated methods for measuring ETS exposure in hair.

**THE ABIS STUDY**

Data supplied by the participants in the ABIS study (All Babies in Southeast Sweden) has been used for study IIIc–VI. ABIS is a prospective, longitudinal, cohort study aiming to study environmental factors affecting development of immune-mediated diseases in children (figure 2). The cohort comprises 17 055 / 21 700 (78.6%) of the children born in the South-east region of Sweden.
(Östergötland, Kalmar, Jönköping, Kronoberg and Blekinge county). The children were included at birth between 1st October 1997 and 30th September 1999. Biological samples were taken at birth (cord blood, breast-milk, mother’s hair), and from the child at 1 year (blood, hair), 2.5 years and 5 years (blood, urine, stool, hair) of age. The parents have also responded to comprehensive questionnaires, the first about circumstances during pregnancy, and the second, a diary, which the parents were asked to keep during the child’s first year. In this they were supposed to make notes on breast-feeding, other nourishment, infections and other illnesses or injuries and immunisations of the child. The parents were also asked to tell if any serious life events had occurred in the family during the year. When the child was one year old a new questionnaire was given to the parents. The questions on nourishment continued, home environment was surveyed and parents’ feelings about parenthood, stress and working conditions were queried (78).

A new questionnaire was delivered at the next checkpoint, which was when the child was 2½-3 years old. The questions were repeated in almost the same format as before: home milieu, parent’s working conditions, eating habits of the child, the health of the child and parent’s feelings about having children, how confident they were in the role as parent and their feelings for participating in this type of study. These questions were repeated when the children were 5-6 years old, and further questions about smoking behaviour and the child’s sleeping pattern are also added. The prospective study will hopefully be prolonged and follow the children in school ages.

Several studies have been and are performed using the ABIS material. One thesis, Gustafsson Stolt (79) has especially studied ABIS from an ethical perspective. The results indicate that parents were less concerned over research material and screening results than often assumed. Confidentiality and integrity were, however, considered to be important by the participants. It was also stressed that the parents’ informed consent was valid only for studies with the primary aims given from the start of the project.

ETS exposure was one environmental factor included in all ABIS questionnaires from the beginning. The results from the studies of this thesis were supposed to contribute with further detailed data on this subject. A large prospective study on the importance of environmental circumstances for the development of immune mediated diseases, like ABIS, has possibilities to contribute to the knowledge of the ETS exposure role.
Figure 2. The research design of ABIS (All Babies in Southeast Sweden).
TOBACCO SMOKE EXPOSURE AND CHILD ABUSE

Exposing a child to tobacco smoke has in various contexts been assigned to child abuse. Child abuse and neglect includes four distinct conditions: neglect, physical violence, emotional abuse and sexual abuse (80). Besides aggressive acts it also includes neglect, lack of adequate protection and failure to nurture (81). Physical abuse in children has been defined as “any act that results in a non-accidental physical injury by a person who has care, custody or control of a child” (p.29, 82).

According to James Garbarino, professor of Human Development at Cornell University, USA, three conditions are to be fulfilled before any parental act can qualify as child abuse or neglect. They are: 1) there must be a basis in scientific knowledge or among professional expertise that a particular practice is harmful or dangerous to children, 2) there must be a public debate going on using the new knowledge as a basis for challenging what has been regarded as normal child rearing, 3) community values must be adapted to the acceptance of a new standard of care for children. Normal conditions first become unwise, then only acceptable and finally illegal (In 83).

To expose a child to ETS has been regarded as child abuse, however, not with the same dramatic features as physical damages, but fulfilling the above mentioned criteria (84). WHO (85) has, in releasing the report “Tobacco and rights of the child”, taken a clear stand and recommended countries to take all necessary legislative and regulatory measures to protect children from tobacco and the tobacco industry. Focus in this report is primarily on children’s own smoking but children’s ETS exposure and child labour in the tobacco industries are also emphasised. The Convention of the rights of the child (80), consisting of legally binding international obligations, and signed by all but two countries in the world, was recommended by WHO to be used as support.

A social norm has come up in society, stating that the act of exposing someone to tobacco smoke needs to be construed as a rude and offensive act, sometimes perhaps a minor assault. In USA, State court has found it appropriate to consider parental smoking in the presence of a child when determining parent custody and visitation (83).
**THE PROTECTION MOTIVATION THEORY**

Protection motivation theory (PMT) created by Rogers (86, 87) was selected as the conceptual framework for showing variables of importance for smoking parents ability to protect their children from ETS. This model proposes that when environmental or personal factors are a threat, like ETS to children's health, decisions regarding coping responses to this threat are made as a result of balancing the costs and benefits of the threats with those of the protecting behaviour. Threat assessment includes evaluating the child's vulnerability to and severity of the threat as well as the intrinsic and extrinsic rewards of experiencing the threat. Protecting behaviour includes evaluation of response efficacy, the perceived likelihood that the using of precautions will reduce the threat, one's own self-efficacy, and the costs and benefits of the precautions taken.

Threat appraisal has 2 parts, the parents' perception of:
- The child's vulnerability: How big is the risk that my child's health will be influenced by ETS exposure?
- The severity of the diseases that might affect the child.

Coping appraisal has 4 parts, the parents' perception of:
- Response efficacy: Are the precautions effective enough to protect my child from ill health due to ETS exposure?
- Their self-efficacy: The parents' belief in their capability to use effective precautions
- Response costs: The trouble and inconvenience that come with the use of precautions, e.g. be cold and wet outdoors, have to leave the comfort indoors, have to leave the child alone
- Response benefits: Like having a clean environment in the house or living up to the social norm

A meta-analysis of the literature on PMT (88) concluded that each component of the PMT was significantly related to healthy attitudes and behaviour and it was usable in prevention and health promotion.

The perception of threat is important but if it is not combined with a high self-efficacy it does not produce protection motivation. If so, the information about hazards of ETS on the health strengthens the feelings of fatalism and hopelessness, which obstruct all efforts to make any changes. According to this, the crucial components in a successful intervention to prevent ETS exposure of children in the homes can be summarised as:
- The parents need to have full information about the known health effects of ETS exposure
- The parents need to know if the precautions they make are effective
- The parents need encouragement to sustain adaptive coping.
Protection Motivation Theory
Rogers RW (86, 87, 89)

**Vulnerability**
How vulnerable is MY child to the threat?

**Severity**
How severe are the health effects that might be the result of ETS exposure?

**Response effectiveness**
How much less is the risk for health effects if I make this/these precautions?

**Self-efficacy**
How capable am I to carry out effective precautions?

Response costs: How inconvenient are the measures?

Response benefits: E.g. a clean house, living up to the expectations of others

Behavioural intentions

Behaviour

The social norm*

Figure 3. The Protection Motivation Theory adapted to children’s passive smoking. The figure is modified for this thesis. Response costs and benefits are included. The social norm* is added by the author.
INCITEMENTS FOR THE STUDIES

My many years of experience as a child health care nurse along with my public health studies made me curious on the meaningfulness of parts of the preventive work in child health care. How well-founded were all the advises we gave to parents? My work with the project “Smoke-free children” led me into the “arena of passive smoking”, and this generated several questions.

The speculations and discussions led to the following questions:

- Does having children affect adult smoking or smoking behaviours in the home? (II)
- Which measures are taken in the home to protect children from tobacco smoke exposure? (I, II)
- Is it possible to determine the effectiveness of the protective measures by means of analysing cotinine in the children’s urine? (III, IV, V)
- Does strictly outdoor smoking prevent children’s tobacco smoke exposure and/or adverse health effects? (I, IV, future studies)
- How are perceptions and attitudes to smoking and passive smoking among non-smoking and smoking parents of young children? (VI)
- How have parents experienced the handling of the smoking issue in health care? (VI)

The discussions and genesis of the research questions are illustrated in Figure 4.
Figure 4. A figure illustrating the research questions.

*the importance for children’s health of different ETS doses, due to the use of different protective measures, has not been shown.
AIMS

The general aim of this thesis was to contribute to a basis for well-founded advice to smoking parents on how they should protect their children from ETS exposure.

It can be divided into the following questions: How common was smoking among parents of pre-school children? Which measures do smoking parents usually take to protect children from tobacco smoke exposure and how effective are they? How was parents’ attitude to smoking and children’s passive smoking and their opinions on tobacco prevention in health care?

The specific aims of the different papers were the following:

- **Determine the prevalence and nature of smoking among parents of infants during their first two years of life, with special reference to indoor/only outdoor smoking. Smoking behaviour was related to socio-demographic background and to the health of the infants. (I)**

- **To study whether having children affects adult smoking prevalence and/or smoking behaviours in the home and how much importance survey subjects placed on protecting the indoor environment from ETS. (II)**

- **To develop and validate an instrument, measuring children’s ETS exposure, with focus on parents’ use of different protecting strategies. (III)**

- **To examine the effectiveness of parents’ modes of action in the homes for limiting their children’s ETS exposure and to identify variables of importance for parents’ choice of smoking behaviour (V)**

- **To increase the comprehension on perceptions and attitudes to children’s ETS exposure among parents of pre-school children and how their smoking and/or smoking behaviour was influenced. Further to increase the understanding of how the parents have experienced the handling of the tobacco issue in antenatal and child health care. (VI)**
**SUBJECTS**

An outline of the subjects in the different studies is presented in *figure 5*. The samples are described in *table 2*.

**Study I**
The participants were sampled from the national registration of Sweden including all children aged 12-24 months (n=1990) born between April 1st 1994 and March 31st 1995 in four municipalities in the county of Östergötland, Sweden. The municipalities represented both rural and urban populations with a mixture of blue-collar workers, civil servants and academics.

**Study II**
Data from a cross-sectional randomised survey, made in the county of Östergötland, Sweden, in 1999 was used. The random sample comprised 10 000 adults, 20-74 years old, collected from the National Registration of Sweden in 1999. After two reminders the participation rate was 65%, with 63% usable for analysis. The sample was analysed with respect to age, sex, having dependent children, immigrant background, marital status, unemployment, education and smoking habits. The result is to be considered in accordance with a randomised group, with the exception of young men and the variable marital status (90).

The sample for this study was the 20-44 year-olds who had stated that they were willing to answer further questions (n=1735). They got the tobacco questionnaire by mail and 78% (n=1352) responded, and created the study population. The sample was compared to the randomised sample of 10 000 and the general Swedish population and was regarded to be representative according to socio-demographic variables (Paper II, table 1).

**Study III**

a) Smoking parents of pre-school children recruited by nurses at CHC clinics, (n=79)

b) A convenient sample of personnel and patients at a Public Dental Health services office. (n=8)

c) Families with a 2½-year-old child participating in ABIS.
   Cases: Families where smoking was reported (n=153)
   Controls: Families where no smoking was reported (n=309)
Study IV
The study object was one family with a 2½ –3 years-old child, participating in ABIS.

Study V
Families with 2½ –3 years-old children participating in ABIS (n=1120). Cases: families where smoking was reported (n=687). Eighty four percent (578/687) responded to the questionnaire and of these 366 also had delivered a urine sample Controls: age-matched children in families from non-smoking homes (n=433) The sample from study III c was included. The final study sample comprised 799 (366 cases and 433 controls) families.

Study VI
Three hundred families who had participated in study V. One hundred families were randomised from the 286 outdoor-, from the 191 indoor-smoking families and 100 from the 433 non-smoking families. Ninety one percent responded (n=272). Some families were recategorised due to changed habits and 9 questionnaires were unusable. Finally 92 non-smoking, 81 outdoor smoking, 82 indoor smoking families and 8 smoking families with unknown behaviour constituted the study sample.
<table>
<thead>
<tr>
<th>Study</th>
<th>Participants in the different studies.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study I</td>
<td>1990 (all in a geographical area)</td>
</tr>
<tr>
<td></td>
<td>1600 (81%)</td>
</tr>
<tr>
<td></td>
<td>12–24 months</td>
</tr>
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<td></td>
<td>All parents</td>
</tr>
<tr>
<td></td>
<td>Smokers and non-smokers</td>
</tr>
<tr>
<td>Study II</td>
<td>1735 (random sample)</td>
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<tr>
<td></td>
<td>1352 (78%)</td>
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<tr>
<td></td>
<td>0–19 years</td>
</tr>
<tr>
<td></td>
<td>Parents and non-parents</td>
</tr>
<tr>
<td></td>
<td>Smokers and non-smokers</td>
</tr>
<tr>
<td>Study III</td>
<td>Smokers parents recruited from CHC</td>
</tr>
<tr>
<td></td>
<td>n=34 (46%)</td>
</tr>
<tr>
<td></td>
<td>2½–3 years</td>
</tr>
<tr>
<td></td>
<td>All parents</td>
</tr>
<tr>
<td></td>
<td>Smokers</td>
</tr>
<tr>
<td>Study III</td>
<td>Smokers at a dental clinic</td>
</tr>
<tr>
<td></td>
<td>n=8</td>
</tr>
<tr>
<td></td>
<td>Parents and non-parents</td>
</tr>
<tr>
<td></td>
<td>Smokers</td>
</tr>
<tr>
<td>Study III</td>
<td>A sub-sample from ABIS (n=462/17 055)</td>
</tr>
<tr>
<td></td>
<td>153 (83%)</td>
</tr>
<tr>
<td></td>
<td>+ 309 controls</td>
</tr>
<tr>
<td></td>
<td>Parents</td>
</tr>
<tr>
<td></td>
<td>Smokers and non-smokers (controls)</td>
</tr>
<tr>
<td>Study IV</td>
<td>1 child (ABIS)</td>
</tr>
<tr>
<td></td>
<td>2½ year</td>
</tr>
<tr>
<td></td>
<td>Parents</td>
</tr>
<tr>
<td></td>
<td>Smokers</td>
</tr>
<tr>
<td>Study V</td>
<td>Sub-sample from ABIS study</td>
</tr>
<tr>
<td></td>
<td>366/678 responded + urine sample + 433 controls</td>
</tr>
<tr>
<td></td>
<td>2½ – 3 years</td>
</tr>
<tr>
<td></td>
<td>Parents</td>
</tr>
<tr>
<td></td>
<td>Smokers and non-smokers (controls)</td>
</tr>
<tr>
<td>Study VI</td>
<td>Sub-sample from study V (n=300)</td>
</tr>
<tr>
<td></td>
<td>272 (91%)</td>
</tr>
<tr>
<td></td>
<td>3½–5 years</td>
</tr>
<tr>
<td></td>
<td>Parents</td>
</tr>
<tr>
<td></td>
<td>Smokers and non-smokers</td>
</tr>
</tbody>
</table>

Table 2. Participants in the different studies.
I
All children 12-24 months old in 4 communities, n=1990

II
Randomised general population in Östergötland
20-44 years old n=5030
Sub sample: n=1735

III
a) Smoking families in CHC (n=79)
b) Convenience sample (n=8)
c) Sub sample from ABIS (n=462)

IV
One family from the sample of IIIc

V
A sub sample from ABIS, IIIc included (n=1120)
687 smoking families:
   respondents 578/687;
   urine sample 366/578
433 controls
Final study sample: n=799

VI
Families randomised from Study V:
n=272/300
   92 non smokers
   81 outdoor smokers
   82 indoor smokers
   8 smoker unknown behaviour
   9 unusable n=263

Figure 5. Samples for the different studies
METHODS

The first studies (I, II) of this thesis were cross sectional and had a descriptive design. Study V and partly study III, were cohort studies with an observational analytic design. Study VI was a cohort study with a descriptive design. Postal questionnairies were used for most of the data collection.

Study I
Study I was a cross sectional survey with a questionnaire developed for the purpose of this study. It was pre-tested in a pilot study and sent to the parents of each child to be answered by either of them. For those who did not answer after four weeks, a new questionnaire was posted.

The questionnaire comprised 32 questions providing information about socio-demographic data, smoking habits of the parents during pregnancy and infancy, attitudes to smoking, child exposure to ETS and the health of the child. The questionnaire had yes/no and open-ended questions as well as VAS scales for opinions on how important it was considered by the parents to be smoke-free during breast-feeding period and during the first two years of the child’s life. They were also asked to state how socially accepted their smoking was considered. Smokers included daily as well as occasional smokers. Three groups of children were compared: children with non-smoking parents, children with exclusively outdoor smoking parents and children whose parents stated that they sometimes smoked indoors.

Parents’ membership in different trade unions was used to characterise socio-economic background. Health variables asked for were conditions known to be related to ETS exposure, e.g. otitis media, wheezing when having or not having a upper respiratory infection (URI), prolonged colds, coughing at night, or coughing more than 2 weeks after an URI.

Study II
Information from a cross-sectional randomised survey in Östergötland, Sweden was used. This instrument consisted of seven domains dealing with demographic issues (n=7), perceived health (n=42), lifestyle (n=34) and both physical and mental health at home (n=27) and at work (n=44). The instrument was composed of well used and tested questions as well as validated instruments like the generic instrument 36-item short form (SF-36) measuring self perceived health related quality of life (90, 91). For this study socio-demographic data, smoking status, parenthood and self-perceived health related quality of life was relevant.
A supplementary questionnaire on smoking behaviour, attitudes to keep the environment smoke-free and which measures taken by the smoker to protect the environment were considered to be effective, was developed, and tested by a convenience sample. The questionnaire was mailed to the sample of this study.

Study III
The development of the smoking behaviour instrument is described. The questionnaire was made in 7 consecutive steps. The first draft was made on the basis of knowledge obtained in Study I and II, and the interviews described in Study VI. Core elements, according to Jarvis (56), for the instrument were identified: number of smokers in the household; cigarette consumption in the home weekdays and weekends; for how long consumption level and/or smoking location had been unchanged; how often and which strategies for ETS protection had been used; how important it was considered by the smokers to smoke in different places and how often the child was exposed to ETS outside the home. The items were scrutinised and commented on by experts on tobacco issues and questionnaire making, by the members of the project group and by some smokers.

Two pilot tests were performed. The first one was made to test content and face validity. Families including at least one smoker and a pre-school child responded to the questionnaire. Reliability test was made with a test retest. The families were asked to respond to the questionnaire twice at intervals of 2 weeks.

After reviewing the results of the first pilot test and discussions with an expert panel leading to some revisions of the questionnaire, the second pilot test was performed. A convenience sample, 8 smokers at a Public Dental Health services office, patients as well as personnel, responded to the revised questionnaire. Within a week they were all telephone interviewed about how they had interpreted the different items. The interviewer was familiar with tobacco issues but not involved in the construction of the questionnaire. The respondents had not been included in any of the earlier tests.

The second pilot test led to some minor alterations and in the last step the questionnaire was validated with biomarkers. The final version of the smoking behaviour instrument (appendix 2) was tested by a sub-sample of the families in ABIS when the children were 2½–3 years old and if smoking was reported in the ABIS questionnaire.
The instrument was sent by post. Urine samples had in most cases been delivered together with the responding of the ABIS questionnaire, i.e. before the parents had received the smoking behaviour questionnaire. A reminder was sent out after about 4 weeks to those who had not responded.

Two age matched children per each case were chosen as controls (n=309). These were children included in the ABIS study whose parents had denied smoking neither by themselves nor by others in the home, when responding to the ABIS questionnaire. A urine sample from these children was also available.

Urine cotinine analyses were performed to test if the instrument could discriminate children's level of tobacco smoke exposure when related to parents smoking behaviour. The cotinine analyses were made with capillary gas chromatography, 'Method NM-018-8' by Pharmacia, Sweden. The analyses were made together with control samples with known values. Lowest Level of Quantification (LLQ) was based on the standard curve and was 6 ng/ml. All children with lower levels got the value '1' in the calculations. Creatinine was analysed on all samples ≥ LLQ to adjust for the dilution of the urine. Cotinine-to-creatine ratio (CCR) was determined as μmol cotinine/mol creatinine.

**Study IV**

A case study in which a child, included in IIIc, with an extremely high CCR level is described. The CCR value was related to the smoking behaviour and cigarette consumption in the home. The CCR value was recalculated into μg cotinine/1 g creatinine, which then was related to the estimation of how much the active smoking of 1 cigarette contributes to the urine CCR level (92).

**Study V**

A cohort study in which the questionnaire and urine specimen from the 2½-3 year-old children in ABIS (figure 2) was used. A sub sample of children whose parents had reported smoking either by themselves or by others in the home (n=687) were sent the questionnaire on smoking behaviour (appendix 2) immediately after the ABIS questionnaire was received. A reminder was sent out after about 4 weeks to those who had not responded.

The families were categorised in 6 smoking behaviour groups, which were used in the analyses of the results. The groups were defined as:

*Outdoors + change:* All smoking in the home was performed outdoors with the door closed and the smoker always changed clothes afterwards.
*Outdoors, door closed:* All smoking was performed outdoors with the door closed

*Open door, outdoors:* The smokers either smoked near an open door or outdoors with the door closed.

*Kitchen fan, outdoors:* The smokers either smoked close to the kitchen fan or outdoors with the door closed

*Mixers:* The smokers either smoked close to the kitchen fan or near an open door or outdoors with the door closed

*Indoor smoking:* All who stated that smoking sometimes occurred at dinner table and/or the TV set and/or anywhere indoors and/or other places indoors. This behaviour was sometimes combined with using the precautions listed above.

The urine specimens provided by the children were analysed for cotinine and creatinine. Cotinine analyses were performed with capillary gas chromatography, ’Method NM-018-8’, by Pharmacia, Sweden. The system was calibrated at 6 levels with 2 replicates at each level. Optimal curve fit was obtained by weighted linear regression analysis. The standard curves were linear in the measured range between 6 and 1200 ng/ml. Results with a concentration lower than the lowest calibration standard were reported as LLQ. The less reliable values, between 2 and 6 ng/ml, were used in some analyses.

Creatinine was measured on all samples $\geq$LLQ to correct for the dilution of the urine. These analyses were performed at Division of Clinical Chemistry, Linköping University Hospital.

The level of cotinine was calculated as $\mu$g cotinine/mol creatinine, and related to cigarette consumption and smoking behaviour of the parents.

Controls were age-matched and chosen among children included in ABIS whose parents had denied smoking and smoking ever occurring in their homes. Cigarette consumption levels were recategorised into none, sporadic, 1–10, 11–20 and >20 cigarettes/day, to avoid small numbers of cases in extreme groups. The answers about children’s ETS exposure outside the home were analysed by content and categorised as 'visiting grandparents', 'visiting friends', 'café and restaurants', 'father’s home', 'outdoors' or 'other'.

*Study VI*

A questionnaire (appendix 1) was made from the basis of 11 semi-structured interviews with smoking parents of pre-school children. The parents were asked to specify to what extent they agreed or disagreed to 23 statements from these interviews. A 5-grade scale of Likert type (i.e.: 1=strongly agree, 2=agree,
3=undecided, 4=disagree, 5=strongly disagree) was used. The statements were grouped in 6 categories: “General attitude to smoking and smokers”, “The influence of ETS exposure on the children”, “How can children be protected from ETS”, “How the issue is handled in antenatal care”, “How the issue is handled in CHC”. Two statements did not fit in the categories and were reported separately. The average response score for each category of statements was calculated for each respondent. A lower score indicated a more smoking-friendly attitude and less knowledge and concern for passive smoking. If an issue had the reverse wording it was recoded.

The questionnaire had also 3 open ended questions in which the parents were asked to comment on how the tobacco issue should be handled in antenatal and child health care. They were also asked to report if the mother or father, respectively, smoked 'daily', 'every week', 'seldom' or 'never', and if the mother, the father, or mother and father together had responded to the questions. There was room for additional comments. Socio-demographic data on the parents were available from the ABIS questionnaire.

Two reminders were sent out to non-respondents. The first after about 4 weeks, and the second after additionally 5 weeks.

**Statistics**
Analyses were performed using the SPSS® Versions 9–11 (SPSS Inc. Chicago IL, USA). Descriptive analyses were used to describe the samples. Open-ended questions were analysed by content and categorised.

One way ANOVA (Analysis of variance), with Bonferroni as posthoc test, was used to show equalities and differences between groups. When data was not normally distributed, the chi-squared test and Mann-Whitney U test were used. When the assumptions for chi squared test not were fulfilled Fisher's Exact Test was calculated.

Pearson’s and Spearman's correlation test and Kendall’s tau-b were used to show correlation between variables.

Multivariate analyses (The Mantel-Haenszel Relative Risk, logistic regression and linear regression models) were performed in order to clarify the association between variables. Dependent variables were dichotomised: urine cotinine level as above or below LLQ (6ng/ml) and smoking behaviour as smoking indoors or outdoors. The strength of the associations was expressed by odd ratios (OR) with 95% confidence intervals (CI).
The cotinine to creatinine ratio (CCR) expressed as micrograms per mol creatinine (µg/mol), the natural logarithms of the CCR values or cotinine values were used in comparisons.

A p-value <0.05 was considered as significant.

**ETHICAL CONSIDERATIONS**

Permissions for the studies were obtained from the Regional Ethics Committee for Human Research at Linköping University (for ABIS also from the corresponding Committee in Lund). The studies have been conducted in accordance with the Declaration of Helsinki. Ethical considerations regarding autonomy and the risk of intruding on someone’s integrity were made in all studies. All participants were sent written information together with the postal questionnaires. It was made clear in all information that participation was voluntary, a telephone number was always given for questions and confidentiality always guarantied. Returning of a completed questionnaire was considered as having given informed consent.

The participants in ABIS (Study III–VI) were given information about participation in ABIS when they were attending antenatal care, after delivery and recurrent information when contributing with data. The information has been given personal, both verbal and written, and general as TV-advertising, newspaper advertising and posters.

When the parents were asked to participate in the extended part of ABIS on smoking behaviour they were ensured that the issue was connected to the primary aims of ABIS and that their participation was voluntary. Smoking was one of the environmental issues associated to the development of immune-mediated diseases, already asked for in the ABIS questionnaires. The added questionnaire and analyses would be a complement contributing to more valid data on the child’s ETS exposure. It was also made clear that all information from parents was confidential and that data was made anonymous as soon as it was received.

In a prospective study like ABIS the identity of the respondents must be known. The identity was coded and the key for code numbers has been safely stored.
well apart from other data and with access only for those researchers who need it.

The ethical considerations on prospective, longitudinal screening have been studied by Gustafsson-Stolt (79) using the ABIS material. The results of these studies indicated that participants were less concerned over research material and screening results than often assumed but expressed concern on confidentiality, integrity and restrictions. They also stressed that the material should not be used for other studies than those originally informed about and to which they had given their informed consent.

The question of smoking and especially when related to children’s passive smoking has become a delicate issue in society today. However, the high response rate to the different postal questionnaires might be looked upon as a sign of interest from participants and show that against that questions were not considered to be offensive, insulting or impertinent. On the contrary we have received many comments on the importance of the studies – both from smokers and non-smokers.
RESULTS

Indoor and outdoor smoking: Impact on children’s health (I)

Smoking prevalence among the parents of 12-24 months old children, was 14% (both for mothers and fathers), and 20% of the children had one or two smoking parents. It was shown that being a young parent (<26 years of age), a blue-collar worker and/or a single parent increased the risk for being a smoker as well as for being an indoor smoker. Eighty-eight percent (52/59) of the mothers who had stopped smoking during pregnancy had resumed smoking at the time of the questionnaire, i.e. before the child was 2 years old. Six percent (n=13) of the smoking fathers stopped smoking during pregnancy and 4% (n=9) had started again before the child was 6 months old.

Smoking behaviour was grouped into smoking only outdoors and indoors and the prevalence of respiratory symptoms like coughing and wheezing, frequency of colds and media otitis (according to parents’ reports) was related to their parents’ smoking status; non-smoking, outdoor or indoor smoking. A trend for an increased prevalence of symptoms related to increased ETS exposure was seen (figure 6). The prevalence of otitis media and pooled respiratory symptoms was also related to smoking behaviour of only daily smokers (figure 7).
Figure 6. Prevalence of otitis media, prolonged colds, cough more than two weeks after URI (upper respiratory infection), wheeze with URI, wheeze with no URI, cough at night, pooled respiratory symptoms (i.e. wheeze with and without URI, coughing at night and more than two weeks after URI) related to smoking behaviour of the parents (percent).
Figure 7. Smoking behaviour of parents (only daily smokers included) related to symptoms of the children (percent). Differences between groups NS.

When the data was stratified according to trade union membership the trend for increased prevalence of disease between smoking related groups remained in most comparisons, though some of the groups became small.

**Does having children affect adult smoking and behaviours at home? (II)**

The focus of this study was to find out whether having children affects adult smoking prevalence and/or smoking behaviour in the home. Smoking prevalence was 31% (occasional smokers 14% included). Parents with preschool children (27%) smoked significantly less than parents with only school children (36%), but to the same extent as adults with no dependent children (30%). Short education, female gender and a low score for mental health (in SF-36) was associated to smoking.

Parents with preschool children (72%) and parents with school children (64%) smoked significantly more outdoors than adults without dependent children (50%). Being an immigrant and not having dependent children were variables associated to indoor smoking.
Smoking parents with preschool children (OR 1, reference) found it significantly more important to protect indoor environment from ETS than non-parents (OR 0.4 CI 0.2–0.6, p<0.001).

Smoking parents compared to non-smoking parents showed lower scores in several domains in self-perceived health-related quality of life measured with SF-36; general health (p<0.01), vitality (p<0.01), social functioning (p<0.01), role-emotional (p<0.01), and mental health (p<0.01). The differences remained when including only parents with preschool children. The variable mental health was significantly associated to both smoking (OR 0.99 CI 0.97–1.00, p<0.05) and smoking behaviour (OR 1.03 CI 1.01–1.05, p<0.01).

Assessment of smoking behaviours in the home and their influence on children’s passive smoking: development of a questionnaire. (III)

The development and validation process of an instrument, measuring children’s exposure to ETS in the home, is described. A first draught was made considering common strategies for protecting children from ETS in the home, established in previous studies (I, II), and core elements for the instrument identified (56). Experts on smoking issues and experts on questionnaire making tested face validity and content validity.

Pilot test I
Smoking parents (n=34) recruited by CHC nurses, tested the first version. Cigarette consumption on weekdays and on weekends were stated. All alternatives for smoking location were used and frequency was reported respectively. The spaces for own suggestions were sparsely used.

A test-retest was performed to show the reliability of the instrument. All families (n=34) got the questionnaire twice at intervals of two weeks. It was possible to match 15 of the answers. A correlation between answers was calculated for the 12 items: $r = 1.0$ (4 items), $r = 0.7–0.9$ (7 items) and $r = 0.5$ (1 item). The differences between the first and second answers were mainly due to different interpretations of “home-milieu”. This was obvious when the comments made in the questionnaires were considered. Two respondents had also differed one step in the frequency table between the answers.

Kendall’s tau-b (0.7, p<0.001) was calculated for the entire instrument (this version)
Pilot test II
The instrument was revised according to deficiencies shown up in pilot test I and distributed to 8 smokers at a Public Dental Health Services office. They were telephone interviewed within a week on how they had interpreted the questions.

No one had found it difficult to understand “home-milieu” when “including balcony, terrace, etc” was added. The alternatives for cigarette consumption and smoking behaviour were regarded as relevant. Several respondents wanted to add “Where?” in the question on ETS exposure outside the home.

The final questionnaire (appendix 2) was designed after consideration of the results from the pilot studies.

Validation with biological markers
Smoking parents of 2½–3 year old children participating in ABIS responded to the final version (n=153). The response rate (83%) indicated that the instrument was easy to understand and not intrusive.

According to the parents’ smoking behaviour 5 exposure groups were made; outdoors with the door closed (52%), exclusively near the kitchen fan (3%), exclusively near an open door (3%), mixing kitchen fan, open door and outdoors (29%), and indoor smokers (9%). Age-matched controls (n=309) with non-smoking parents who stated that smoking was never occurring in their home, were chosen among the “ABIS-children”. Urine cotinine and creatinine analyses were performed and the CCR was calculated for each child. The children’s CCR was related to the smoking behaviour groups, 4 groups were big enough to be included in the analyse (figure 8).
When does exposure of children to tobacco smoke become child abuse? (IV)

In this letter we wanted to point out that a child of 2½ years of age, still might live in an exceptionally ETS polluted milieu. The child had a CCR of 800 µg cotinine/1 g creatinine, corresponding to the active smoking of 3-5 cigarettes per day (92). This value was much higher than the values of any of the other children in the study sample for study V.

The cigarette consumption in the home was among the highest in the study (41-60 cigarettes/day) and the smoking behaviour was reported to be fairly unrestricted. The parents reported smoking at dinner table and the TV set several times a day. Measures of protection used in the home were smoking near the kitchen fan and near an open door. However, comments in the questionnaire indicated that the parents regarded their children to be satisfactory protected since no smoking was performed in the bedrooms and the windows were almost always open.
How should parents protect their children from ETS exposure in the home? (V)

The effectiveness of commonly used measures of protection taken by smoking parents in the home in order to protect children from ETS exposure was examined. The developed and validated questionnaire on smoking behaviour (Study III) was responded to by smoking parents of 2½-3 years old children included in ABIS (578/687, 84%). From 63% (n=366) of these children an urine sample was available and cotinine and creatinine analyses were performed. Controls (n=433) were age-matched and chosen among ABIS children whose parents had denied smoking by themselves as well as by visitors in the home. No difference in mean CCR was seen between boys and girls.

More than half of the parents stated that they always smoked outdoors with the door closed and very few smoked anywhere indoors, at the dinner table or when sitting at the TV-set (figure 9). The parents smoking behaviour was related to the CCR (figure 10) and cotinine values (all =2 ng/ml included) (figure 11) of the children. The small groups “always smoking outdoors + change clothes” (n=10), “exclusively smoking near the kitchen fan” (n=9) or an “open door” (n=7) were added to groups combining this with “outdoor with closed door” smoking.

Figure 9. Distribution of families according to smoking behaviour. Number of families.
Figure 10. Mean log CCR related to smoking behaviour

Figure 11. Smoking behaviour related to mean cotinine values
All values ≥ 2 ng/ml included.
The parents were asked for how long time their cigarette consumption and smoking behaviour had been in the described way and 91% of the families had had unchanged conditions for 12 months or more. It was obvious that the main part of children’s ETS exposure took place in the home since 86% of the children were never or seldom ETS exposed outside the home. If it occurred the most common place was when visiting grandparents (46%).

Multiple logistic regression models showed that high cigarette consumption and not living in a nuclear family was of importance for choice of smoking behaviour. Smoking behaviour was of importance for ETS exposure.

An exposure score illustrating the effect of different smoking behaviours was calculated. A logistic regression model with cotinine dichotomised as < or > LLQ as dependent variable and smoking behaviour as independent and categorical variable was made. Score levels are given as odds ratios in table 3.

<table>
<thead>
<tr>
<th>Smoking behaviour</th>
<th>Score</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls (n=433)</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outdoors, door closed* (n=216)</td>
<td>1.99</td>
<td>1.1- 3.6</td>
<td>0.015</td>
</tr>
<tr>
<td>Open door + outdoors (n=45)</td>
<td>2.39</td>
<td>0.9- 6.1</td>
<td>0.069</td>
</tr>
<tr>
<td>Kitchen fan + outdoors (n=50)</td>
<td>3.23</td>
<td>1.3- 7.9</td>
<td>0.010</td>
</tr>
<tr>
<td>Mixers (n=27)</td>
<td>10.32</td>
<td>4.3- 24.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Indoor smokers (n=28)</td>
<td>15.09</td>
<td>6.6- 35.3</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

* 'outdoor +change’ (n=10) here included in 'outdoors, door closed'

Table 3. Exposure score for different smoking behaviours, 95% CI and p-values.

Though cigarette consumption did not influence cotinine values in the logistic regression models it was significantly correlated to smoking behaviour ($r_s=0.23**$). The higher cigarette consumption the more sparsely awkward measures of protection were taken (figure 12).
This study was carried out to obtain a better understanding of attitudes and opinions to children’s passive smoking among parents of pre-school children and how their smoking behaviour was influenced by these attitudes. Three groups with 100 non-smokers, outdoor and indoor smokers were asked to agree or disagree to statements in a postal questionnaire (appendix 1). After two reminders 272/300 (91%), equally distributed between the three groups, had responded. After having considered changed smoking status in some cases the sample comprised 92 non-smoking, 81 outdoor and 81 indoor smoking families. Differences in mean score for the six categories of statements between non-smokers, outdoor and indoor smokers are shown in table 4.
<table>
<thead>
<tr>
<th>Category</th>
<th>Non smoking</th>
<th>Outdoor smoking</th>
<th>Indoor smoking</th>
<th>p-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>General attitude to smoking and smokers</td>
<td>4.3</td>
<td>3.6</td>
<td>3.3</td>
<td>A/B&lt;0.001, A/C&lt;0.001, B/C 0.06</td>
</tr>
<tr>
<td>The influence of ETS exposure on children’s health</td>
<td>4.1</td>
<td>3.8</td>
<td>3.5</td>
<td>A/B 0.08, A/C &lt;0.001, B/C 0.02</td>
</tr>
<tr>
<td>The influence of ETS exposure on children’s own future smoking</td>
<td>4.0</td>
<td>3.9</td>
<td>3.8</td>
<td>A/B 1.00, A/C 0.92, B/C 1.00</td>
</tr>
<tr>
<td>How can children be protected from ETS</td>
<td>4.7</td>
<td>4.7</td>
<td>4.0</td>
<td>A/B 1.00, A/C &lt;0.001, B/C &lt;0.001</td>
</tr>
<tr>
<td>How the issue is handled in antenatal care</td>
<td>4.4</td>
<td>3.7</td>
<td>3.2</td>
<td>A/B&lt;0.001, A/C&lt;0.001, B/C=0.003</td>
</tr>
<tr>
<td>How the issue is handled in CHC</td>
<td>3.8</td>
<td>3.2</td>
<td>3.1</td>
<td>A/B &lt;0.001, A/C&lt;0.001, B/C=0.73</td>
</tr>
</tbody>
</table>

Table 4. Mean score for the categories related to smoking status of the families. Differences calculated with One-way ANOVA and Bonferroni posthoc test. Score range 1-5; 1=worst, 5=best.

About 60% of the smokers stated that it was shameful to smoke in the company of a child. A high score on the “Attitude” score was positively associated with smoking but was of no importance for smoking behaviour. A majority of the all the parents (82%) thought that ETS exposure increased the risk for disease among children. However, 41% of both non- and indoor smokers agreed to the statement saying, “They say that tobacco smoke is dangerous for children, but never why”. Outdoor smokers seemed to be better informed. Indoor smokers were least convinced that the adverse health effects of ETS were proven and also tended to think that the information about the risks was exaggerated with the intention to frighten.
Half of the parents, independently if they were smokers or not, did not think that children’s ETS exposure was of importance for if they would take up smoking themselves as adults. There was a significant difference between nuclear/broken families (p<0.001) and Swedish/immigrant families (p=0.02) on how children could be protected from ETS exposure.

Another aim of the study was to increase the understanding of how the parents had experienced the handling of the tobacco issue in antenatal and child health care. About 30% of the parents stated that they had got a good or fairly good support from the midwife and/or the CHC nurse. Half of the parents did not think or were undecided if their smoking was of any concern to the CHC nurse. The parents had few suggestions for how to improve the preventive work. They suggested help for smoking cessation, group discussions and information about how children could be protected from ETS exposure.
DISCUSSION

METHODOLOGICAL ISSUES

Participants
In the first study (I) and for ABIS (III; IV; V; VI) all families in a specified area with a child born during a specified period were invited to be included, thus no selection was made by the researchers. One possible risk is that the participation of non-Swedish speaking families is sparse causing an under representation of this category. In study II the basic population was randomised from a geographical area, however, later in the process other ways of selections might have biased the results. Therefore the socio-demographic data of the study sample was compared to the randomised group (Paper II, table 1). In study III different samples, mostly recruited by convenience sampling, participated. The respondents in study VI were randomised from participants in study V.

All studies are performed in the South-East part of Sweden which might be a weakness. However, the area comprises 1.1 million people and includes both rural and industrial areas and universities. The area represent both sparsely and densely populated districts and includes two of the eight largest cities in Sweden. The area represents several counties (Study I and II only one county, Östergötland) and about 250 CHC units, thus giving a fairly representative sample of the Swedish families with preschool children.

The samples of the studies have all been population based and thus no risk for bias from selecting a group of children with some illness. The children have been in preschool ages, mostly 2-3 years old, which have ascertained that the children could not be active smokers themselves, they lived in proximity to their parents and they depended on them or other caretakers. Further, breastfeeding was most often ended and could not contribute to the children’s cotinine levels. Other studies looking at children’s ETS exposure have focused on asthmatic children (93) or socially disadvantaged groups (58, 94).

Though smoking and passive smoking has been regarded as a delicate issue to ask about the range of response rates in the different studies has been 78-91%. This indicates high acceptance and interest among the participants. The use of trade union membership for socio-economic classification (Study I) has not been very common and might have caused some misclassification. However, other studies (95) have shown that trade union membership gave the same distribution of smokers among social classes as education level. In the following studies (II–VI) education level has been used to characterise socio-economic background.
The characteristics of the dropouts in the studies are mostly unknown. Two plausible explanations are either it has been smokers who felt accused and did not want to answer questions on their smoking behaviour or it has been non-smokers who did not think smoking was of their concern at all. Two possibilities influence results in different directions.

Methods
Epidemiological methods are the traditional and primary choice when studying a public health issue like smoking and environmental epidemiology is the study of the impact of the environment on human health in populations (53). The two first studies (I, II) were descriptive and from the results hypotheses were formulated. These hypotheses were tested in study V and VI. To determine a causative relationship it will be necessary to use prospective data and the ABIS cohort will hopefully make it possible to follow these children prospectively and thus give an opportunity to show if a causative association between reduced ETS exposure and less ill-health among children can be shown.

Postal questionnaires, developed for the different studies, have been used to collect data. The questionnaires (Study I, II and VI) were scrutinised and pretested, however, not as carefully as in the described process of the development of the smoking behaviour questionnaire (Study III–V). The face and content validity was supported by thorough discussions, reviews and pilot tests.

The truthfulness of the answers can only be speculated on, but probably some underestimation of both smoking prevalence and prevalence of children’s ETS exposure can be assumed. There are studies showing that parents tend to underreport children’s respiratory symptoms especially if the questions are related to smoking (96, 97). This is probably due to a wish to give socially acceptable answers. However, in spite of their weaknesses, questionnaires have been regarded to give a fairly good picture of children’s ETS exposure (56, 58). When parents have been asked about their use of protective measures they might have overestimated their consequent use of them. Hence any of the discussed effects from ETS exposure might have been underestimated.

The process of validating the smoking behaviour questionnaire is described in detail in Paper III. However, it is important to point out that the test-retest is not made on the final instrument. The test led to revisions of the identified weak parts of the questionnaire and no new reliability test was performed on the final instrument.

In studies the results of exposure measurements might have been influenced by the fact that parents are asked about their smoking behaviour and cigarette
consumption. This is probably mostly an unaware process illustrating a wish to avoid criticism (64). In the studies (III, IV, V) the urine sample was delivered before the detailed questionnaire on smoking behaviour was responded to, thus avoiding this possible bias. The aims of the analyses of the urine samples as well as of the other biological samples delivered by the participants in ABIS have been unspecified but strictly bound to the primary intentions of ABIS.

The approach was to ask the parents for the average cigarette consumption in the home and how smoking usually was performed. This might have caused some unexplained high or low cotinine values because of an occasionally high or low ETS exposure prior to the delivery of the urine specimen. The influence of this was minimised with a big sample. The assumption behind this approach was that it would be easier for the respondents to give honest answers and to avoid difficulties in remembering the exact circumstances.

It has been argued that hair nicotine level is a better biomarker than urine cotinine (76). Children from non-smoking, outdoor and indoor smoking homes were compared and they found that hair nicotine levels were better able to discriminate the groups. One advantage is that hair analyses reflect the history of exposure better and no differences in metabolism bias the results. Zahlsen and Nilsen (77) showed already in 1994 that hair analyses are usable to detect ETS exposure level. They pointed out that it is important to cut the hair properly and notice which end has been closest to the scalp. The hair taken from the children in ABIS might be useful, but analyses are expensive and the procedure of hair sampling is not described.

Laboratory methods
The cotinine analyses in these studies were performed with gas chromatography and the performer (Pharmacia, Sweden) specified LLQ to be 6 ng/ml. According to Willers (92) this level corresponds to the active smoking of 0.1 cigarette /day. The high LLQ at Pharmacia was a disappointment. In an earlier pilot study Pharmacia reported LLQ to be 3.8 ng/ml and values as low as 0.9 were given. These new rules changed the assumptions for the study in a way, which was impossible to predict. More sensitive analyses might have given more distinct results and differences between “open door smokers” and “kitchen fan smokers” possible to show. However, we obtained the values below LLQ and used them in some analyses. This was discussed carefully and was done with the reservation that the results should be interpreted with caution. However, even with the high LLQ we have been able to show the important differences between groups of children with different exposure related to smoking behaviour. These are results that would not disappear if a more sensitive method had been used. For further studies, more sensitive methods like liquid chromato-graphic
analyses with spectrometer assays are warranted. If plasma analyses could be used the diuresis does not have to be considered.

Only one urine specimen was delivered from each child and it has been argued that studies of cumulative toxic exposure or long term behavioural patterns require more frequent or continuous measures (64). However, Benowitz et al (20) have concluded that a steady state can be assumed if there is a daily exposure. The ABIS cohort will make it possible to get at least two measurements from each child during preschool ages.

RESULT ISSUES

Distribution of smoking and smoking behaviours
The main results of the first study (I) showed smoking prevalence among parents of 1-2 years-old (14%), consistent with the national statistics (98) and indicated that smoking often is resumed after delivery and breast feeding. Further the results indicated a trend for more symptoms and diseases among children of outdoor smoking parents than non-smoking parents, but less than among children of indoor smoking parents. The relatively small number of smokers might have contributed to the failure to find significant differences between children with outdoor smoking parents, with non-smoking and indoor smoking parents, respectively, and thus caused an occurrence of a ß-error. Further the wording of the questions was not tested as carefully as in the later studies, which might have caused some misclassification of parents. The information about the children’s health was given by the parents, and other studies have shown that parents tend to underreport children’s respiratory symptoms, especially when it is in connection with smoking and ETS exposure (99).

The smoking prevalence and smoking behaviour between parents and non-parents was compared and the results indicated that parents of preschool children smoked to the same extent as adults without children. The smoking prevalence was highest among adults with children in school ages (7–19 years old). The higher mean age of this group (mean 39, SD 4.0) compared to adults with no children (mean 29, SD 6.1) and parents with preschool children (mean 33, SD 5.2) might have contributed to this. Smoking statistics for Sweden show that the age group 35–64 years have the highest prevalence of smokers (98).

However, parenthood influenced smoking behaviour. Results in concordance with Okah et al. (100) who showed that having children was the variable strongest associated to the presence of smoking restrictions in the home.
The differences in health-related quality of life (SF-36) between smokers and non-smokers, as well as ex-smokers has been shown earlier (101, 102, 103). The results of the study, showing lower scores for smokers than non-smokers in 5 out of 8 domains in a group of 20–44 year-olds indicate that smoking cannot be seen as an isolated issue. The period in life with pre-school children is often trying in different ways. Less robust individuals might use smoking as a self-medication to manage the day (104). Mental health had an impact on both smoking prevalence and smoking behaviour but not on “how important it was considered to protect indoor environment”. This might indicate that the ambitions are the same in all groups but the ability to fulfill one’s intentions is less when variables measured in the domain “mental health” get a low score.

Development of an instrument
One of the hypotheses emanating from these descriptive studies (I, II) was if it is possible to determine the efficacy of the commonly used precautions. The first step to test this hypothesis was to develop and validate an instrument. When making an instrument to monitor children’s ETS exposure in the home several variables have to be considered (56). Some of these were already available in the ABIS questionnaires and focus for the new instrument was to survey smoking behaviour in the home, establish average cigarette consumption in the home and get information on the child's ETS exposure outside the home. Further the alternatives for smoking behaviour were given according to earlier studies (I, II) which had shown strategies commonly used among parents of young children in Sweden. The results also gave the impression that most smokers adapt one or two strategies which they use fairly consistently.

The process of development and testing the instrument had its weak parts. The failure to get more answers possible to match for test-retest was probably due to the indirect contact with respondents with no possibility for reminders. However, ambiguous parts were identified in the two pilot tests and clarified, and the instrument has worked well and been responded to by 687 families in study V. The high response rate (84%) might indicate that parents found it important and did not feel accused by the questions.

This questionnaire was, after validation with a biological marker, i.e. cotinine, intended to be usable both in further research, giving extended data on children’s level of ETS exposure, and in clinical situations to create a basis for a discussion. It is in the clinical situation that the questions on the importance of different smoking behaviours are supposed to be used.

How children are protected from ETS exposure.
One child, included in the validation of the questionnaire, had an extremely high CCR level and was described in a letter (IV). The word “abuse” in the headline
might cause objection and must be interpreted in its extended conception in this connection. We are fully aware of the terrible acts that can be referred to by this word, making ETS exposure look trivial. However, as the awareness of the adverse health effects from ETS exposure increases and the norms in society changes, making ETS exposure unacceptable among most adults in society, the exposure of children become a conscious ruthlessness. Different social contexts have different social norms and a parent who knows that ETS exposure is harmful but does not think they smoke much around their child might believe that that level of exposure is trivial. Therefore the question mark in the headline of paper IV has a dual purpose; to question if the awareness of the effects from ETS exposure among parents has become general and to question which “degree of exposure” should qualify for the designation “abuse”.

The regulation stipulated for ABIS says that the families will not be notified on laboratory results if they do not ask for them by themselves. Hence this family was not informed. The situation was neither assigned as child abuse or neglect and thus not an object for report to authorities. The purpose of our report was to draw attention to the fact that children in Sweden still can be extremely exposed to ETS.

To protect the home environment most smokers in Sweden have taken inconvenient measures like standing on windy balconies and opening doors or creeping in under the kitchen fan when they smoke, but the knowledge on the effectiveness of these measures has been scarce. The results of study V indicated that smoking behaviour was of significance for children’s ETS exposure in the home. Almost 60% of the parents stated that they always smoked outdoors with the door closed, which was shown to significantly decrease ETS exposure. Earlier studies have shown that parents’ cigarette consumption was of importance for the children’s exposure (105). The results indicated that level of cigarette consumption significantly influenced smoking behaviour. Increased cigarette consumption caused an increased ETS exposure if smoking behaviour is unchanged, but it also seemed to cause less use of protective measures. By showing this group that it is worthwhile to make some extra efforts to protect the children, it might be possible to improve conditions for the most exposed children.

ETS exposure was related to a detailed description of the average smoking behaviour and the average daily cigarette consumption. Since smoking behaviour seemed to follow a special pattern, with a few, commonly used manners, the parents were asked to describe how their usual smoking behaviour. The results indicate that many parents were very strict and always smoked in the same place. If the parents were less consistent and had more liberal rules for smoking in the home their children were more exposed to ETS and differences
between cotinine levels were evident. This was in concordance with Wong et al (94), who reported differences in cotinine levels between children from homes with absolute, conditional and no restrictions on household smoking, and Wakefield (106) who compared children’s exposure in homes with a complete ban, ban with exceptions, smoking indoors with special rules and unrestricted smoking. Other studies aiming to study children’s ETS exposure in the home have defined smoking behaviour in other ways. Pizacani et al (107) distributed precautions as full ban (no indoor smoking), partial ban (restrictions for time and place allowed for smoking indoors), no ban (smoking allowed anywhere). Callais et al (66) focused on cigarette consumption and asked for number of cigarettes smoked inside the home 2 days before urine collection and did not find their questionnaire able to discriminate between non-exposure and mild exposure. No earlier study has been found showing the importance of different clearly defined measures for protection. Further research is needed to evaluate the effects of reducing ETS exposure for morbidity and mortality (64).

The groups with parents only smoking in an open door (n=9), only near the kitchen fan (n=9) and always changing clothes after outdoor smoking were small in the study, and the protective effect of these behaviours was impossible to evaluate. They were therefore included in the groups combining these behaviours with outdoor smoking. However, it was obvious that any deviation from smoking outdoors was reflected in the cotinine levels of the children and the results indicate that smoking near an open door is somewhat better than smoking near the kitchen fan. Outdoor smoking with the door closed seemed to be effective, though not a total, protection since cotinine levels of these children were significantly higher than of the controls. Changing clothes after outdoor smoking, washing hands after smoking, or staying outdoors for some time after smoking are examples of advice given to smoking parents (108) without any scientific foundation.

Other studies have been performed to examine the relation between reported use of protective measures and children’s cotinine levels (106, 109, 110). The strength of this study was the narrow age range for the children, the population based and fairly large sample, the validated questionnaire with specified strategies for protection and the control group from non-smoking homes. Probably the control group was unusually free from ETS exposure compared to children in other countries; Sweden has a legislation which forbids smoking in most milieus where children could be, therefore resulting in a low prevalence of ETS exposure out of the home. Therefore parents’ measures of protection probably play a substantially higher relative role for children’s ETS exposure in Sweden than it might do in countries where there is a higher “background” exposure. This might limit the generality of the results.
The calculation of scores reflecting the effectiveness of different methods of ETS protection was made with a logistic regression model. This made it necessary to use a dichotomised cotinine variable (< or > LLQ). However, the results reflect the differences seen between the behaviour groups when using both CCR values and cotinine values ≥ 2 ng/ml (figure 10, 11). The score of ETS exposure when using different precautions can be compared to ETS exposure intensity scores calculated in Seifert (111) based on if there were one or two smoking parents, if they smoked in the home and if there were other ETS sources.

The home, as the major ETS source has been described earlier (22, 23) and was obvious in this study since only 12% of the children were in smoky environments outside the home every week and 2% every day. The most common source of ETS exposure was reported to be the grandparents, results in concordance with Hopper (112). This is a generation with higher smoking prevalence (98) and used to more liberal rules for smoking in society. It may also reflect difficulties for the parents to comment unfavourably on the smoking behaviour of this group. Coming legislation will, probably eliminate the second ETS source outside the home, restaurants and cafés.

The smoking behaviour and cigarette consumption was reported to be constant in the families, this was inconsistent with Eriksen et al. (113). They found parent’s smoking behaviour to be labile. The slightly older children of our study might have contributed to this difference.

The knowledge on how ETS exposure influences the health of children and how they can be protected was of importance for how smoking was performed (VI). It was obvious that indoor smokers were less convinced that ETS exposure caused adverse health effects and to a higher extent thought measures like airing the room or “blowing the smoke away from the child” was effective. These findings were compared to Helgason & Lund (114) who, in 1995, found that 51% of smokers and 73% of non-smokers believed in an increased risk for disease. Corresponding figures from this study 7 years later were 51% and 56%, indicating a slow increase of consciousness.

Like in several other studies (114, 115, 116) a short education was correlated to a lower awareness of the risks with ETS exposure as well as a more positive attitude to smoking. The statements used in the questionnaire were taken verbatim from interviews performed in 1996, and regarded to be fairly provocative. However, still after 7 years there were parents agreeing to all of the statements.
Prevention

Though the norms for smoking in society have changed remarkable during the last decade, the results indicate that there is still more to be done. The children of this study sample were born in 1997–1999, which was a period when the nation-wide intervention “Smokefree Children” (14) was implemented in CHC, really putting smoking on the agenda. A similar intervention was initiated in antenatal health care. In spite of this, most of the parents were dissatisfied with the handling of the smoking issue in health care. No activity apart from filling in the health record had been noticed by the parents and only half of the parents thought that their smoking was of any concern to the CHC nurse. This is not to be interpreted as a true description of the nurse’s work but a description on how the parents had experienced the handling of smoking in CHC.

A review (117) of 19 studies describing interventions for reducing residential ETS exposure concludes that such interventions can be effective. However, self reported reductions were shown but only one study found significant differences in cotinine levels between treatment and control group. The importance of interventions based on social cognitive theory and behaviour-modification principles was stressed. The author’s directions for further research describe an intervention very similar to “Smokefree Children”, further they suggest immediate cotinine feed-back for parents, stress management, nicotine replacement therapy and help for weight loss. They also suggest “Motivational interviewing” (118), a client-centred, directive but non-judgemental approach. This method has also been implemented in Sweden, starting with antenatal care and is now being introduced in health care by the National Institute of Public Health.

A review of the effectiveness of interventions aiming to reduce children’s ETS exposure was also made for the Cochrane database (119). Their conclusion was that there was limited support for intensive counselling interventions and they found no difference in effectiveness between different settings for the activity.

Further efforts are also needed to convince nurses and doctors that repeated non-blaming discussions with parents about their smoking are valuable. Tanski (120) showed that the rates of tobacco counselling both at well-child visits and at illness visits for diagnoses directly affected by tobacco use and ETS were extremely low. Winickoff et al (121) also points out that children’s hospitalization are opportunities to influence parents’ smoking and smoking behaviour. Even if a great deal of the work turning the social norm for smoking in society is performed in other arenas (122), it should be complemented in health care. In health care there are so many opportunities for repeated individual, and hopefully, trustful and reassuring meetings. However, the importance of being aware of the delicate character of the issue must be stressed.
To be paternalistic in the context of consultations has not been a successful strategy (123, 124). There are also anti-smoking activists stigmatising smokers as a group, which has become easier when smoking has been the habit of unprivileged groups in society.

In literature there has been a debate on how far it is justifiable to go in the efforts to create an ETS free environment. Repace (125), physist, asserts that non-smokers might be exposed to the same or higher levels of ETS on outdoor sporting arenas and cafés as indoors. Chapman (126) emphasize that as long as science has not shown adverse health effects from occasionally outdoor exposure, the arguments for banning smoking outdoors are rather “aesthetic” than health-founded and should be kept apart. The only consequence is that it infects tobacco control with authoritarian doctrines.

Other possibilities to improve prevention work have been discussed, especially methods to give immediate feedback on the effectiveness of preventive measures taken. Hovell (64) states that the technology needed to develop dosimeters collecting fine particles exists today. The development of an immediate measure of biomarkers is reported to be in progress (127, and personal communication with Cope G, in August 2002) but (to my knowledge) no product, sensitive enough to quantify ETS exposure, is available today. The idea, about giving parents immediate feedback on their measures of recent protection, is promising. It would make parents independent and empowered to deal with their smoking, without having to be questioned by others.

Sweden has the lowest infant mortality in Western Europe and Swedish children consider their own health and quality of life to be good. However, like in other countries there is a socio-economic gradient in children’s health. The socio-economic position of the family has been calculated to explain about 20-40% of children’s ill health in Sweden (128). Since smoking follows the same social distribution, passive smoking is probably one variable contributing to this lack of equality in children’s health. Poulton et al. (129) showed that children’s experience of socioeconomic disadvantage was associated with a wide range of health risk factors and outcomes in adult life and stressed the importance of directing resources towards childhood to improve population health. The goal of the National Public Health strategy for Sweden (130) is that in year 2014, all newborn babies will have a smokefree start to life. To achieve this it will be necessary to find strategies reaching the hardcore smokers (131) and vulnerable groups with short education and immigrant status. The PMT model (87) (figure 4) might be used as a guide for the work. The results of this thesis indicate that further efforts are needed to convince all parents that ETS exposure causes adverse health effects. Further the results might contribute to give an answer to
the variable “Response effectiveness”, though health effects from different measures of protection has not been shown yet.
CLINICAL IMPLICATIONS

An important issue for all health care personnel is to support and empower parents in their parenthood. Smoking, and especially smoking and children, have become delicate issues, not so easy to talk about. A lot of feelings of guilt, defiance, inferiority, possible accusation, hopelessness, being patronised….. – are feared by both parties. However, the main interest and all-embracing goal of both parties of the dialog is to make everything as good as possible for the children.

The instrument developed to survey parents’ smoking behaviour in the home might contribute to overcome these difficulties by helping smoking parents to reflect over their smoking behaviour and how important these behaviours are to them. Their smoking behaviour might also be compared to the calculated exposure score.

RESEARCH IMPLICATIONS

A complement to the performed studies would be a study with a crossover design. The same family would be asked to smoke in a described manner during 2-3 days followed by a non-experimental period of 7 days, and then be asked to smoke in another way, e.g. near the kitchen fan, for 2-3 days. This will be repeated until all the common ways of protection have been used (n=4). After every experimental period a cotinine analyse will be performed on urine or plasma samples from the child to measure exposure and from the parent to measure cigarette consumption. With this design it would be possible to keep several variables, like individual variance in nicotine metabolism, size of dwelling and other circumstances, that random the results, constant. An attempt to perform a pilot study with this design has been made but failed due to difficulties in getting co operative families to carry out all the steps.

From these studies we cannot actually tell the importance of using different precautions for children’s health. We can just speculate from the results in the first study (Study I) which indicated a trend for a dose-response association between exposure level and prevalence of symptoms and Study V where the same dos-response association was seen between subjective exposure level and objective measurements. A remaining task will be to investigate if the differences in exposure level can be connected to differences in prevalence of symptoms/ disease. The ABIS- material will give good opportunities to follow these children with known ETS exposure level prospectively. ABIS also provides data on possible genetic as well as environmental confounders.

An important task is to establish if the difference in exposure between children of outdoor smokers and children of non-smoking families is of any clinical
importance and if/how different levels of ETS exposure influences the development of immune mediated diseases.
WHAT IS NEW IN THIS THESIS?

• Smoking behaviour but not smoking prevalence was influenced by parenthood.
• An instrument for measuring children’s ETS exposure in the home was developed and validated.
• Smoking behaviour of the parents was of importance for children’s tobacco smoke exposure.
• An exposure score was calculated and showed the effectiveness of common precautions used by smoking parents in the home.
• Exclusive outdoor smoking was shown to be the best, though not perfect, way of protecting children from ETS exposure in the home.
• Parents were not satisfied with tobacco prevention in health care.

CONCLUSION

The best way to protect children from ETS exposure, except from smoking cessation, was to perform all smoking outdoors with the door closed. Society, including health care, needs to make further efforts to reach and support groups of parents who smoke indoors.

Syftet med delarbete I, en deskriptiv epidemiologisk tvärsnittsstudie, var att kartlägga hur vanligt det var att barn i åldern 12-24 månader hade rökande föräldrar, belysa rökbeteendet i hemmet samt att relatera prevalensen av symtom/sjukdom hos barnen till föräldrarnas rökbeteende i hemmet. En enkät skickades ut till alla föräldrar i centrala Östergötland som, i mars 1996, hade ett barn mellan 12 och 24 månader (n=1990). Föräldrarna besvarade frågor om boendemiljö, socio-demografiska data, barnets uppfödning och hälsa samt sina rökvanor. Data som bearbetades för denna studie var framför allt frågor om barnets hälsa och föräldrarnas rökning. Resultatet, att även barn med strikt utomhusrökande föräldrar hade en tendens till mer symtom på ohälsa än ”icke rökares” barn, och mindre än ”innerökarnas” barn, genererade följande frågor:

- Påverkas rökprevalens respektive rökbeteende av att man har barn? (II)
- Vilka åtgärder vidtas i hemmet för att skydda barn mot tobaksrök? (II, III, V)
- Avspeglas skillnad i effektivitet hos dessa åtgärder i cotinin analyser av barnens urin? (III, IV, V)
- Skyddar strikt utomhusrökningsbarnen från exponering för tobaksrök? (V)
- Hur ser rökande och ickerökande föräldrars attityder till rökning och passiv rökning ut och hur har de upplevt hälsovårdens tobakspreventiva arbete? (VI)

För studie II användes resultatet från en befolkningsenkät, gjord i Östergötland 1999. Den kompletterades med ett frågeformulär om rökning och rökbeteende, och skickades ut till personer som i befolkningsenkäten angett att de var villiga att besvara ytterligare frågor. Vi jämförde här rökprevalens (tillfällighetsrökare inkluderande) och rökbeteende mellan vuxna (20-44 år) med barn 0-7 år respektive 8-19 år och vuxna utan barn (n=1352), samt hälsorelaterad livskvalitet (enligt SF-36) mellan rökare och icke rökare. Föräldrar med skolbarn (36%) rökte signifikant mer än de övriga grupperna, vuxna utan barn (30%) respektive föräldrar med förskolebarn (27%). Den något högre medelåldern i gruppen med skolbarn kan ha bidragit till detta. Resultatet tydde på att rökbeteendet, men inte rökprevalensen, påverkades av om man var
förälder eller inte. Om man hade barn var man mer benägen att röka utomhus. Kort utbildning, kvinnligt kön och låg score för domänen ”Psykisk hälsa” (SF-36) var associerat till att man var rökare.


I arbete IV beskrivs ett exceptionellt fall som ingick i materialet för delarbete 3. Ett barn, 2½ år gammalt, hade ett cotinin/kreatininvärde som var mycket högre än alla andra barn i studien. Enligt beräkningar motsvarade denna nivå en egen rökning av flera cigaretter/dag. Föräldrarna redovisade en ovanligt hög cigarettkonsumtion i hemmet samt ett, i förhållande till andra familjer i studien, ”fritt” rökbeteende. Avsikten med detta ”correspondence letter” var att visa att barn, trots alla insatser i samhället, även idag, kan vara massivt exponerade för tobaksrök.

I delarbete V beskrivs hur det utarbetade instrumentet skickades till föräldrar med 2½–3 år gamla barn. Familjerna var inkluderade i ABIS studien (Alla Barn i Sydöstra Sverige), en prospektiv kohortstudie som syftar till att belysa miljöfaktorer som har betydelse för utvecklandet av diabetes och andra immunmedierade sjukdomar. Vi vände oss till familjer som i ABIS frågeformulär angett att de var rökare eller att rökning förekom i hemmet. Datainsamlingen pågick mellan april 2001 och januari 2003 och resulterade i att 578/687 (84%) familjer hade besvarat formuläret. Av dessa hade 366 även lämnat ett urinprov från barnen och uppfyllde därmed kriterierna för att ingå i studien. Ytterligare 433 åldersmatchade barn ur ABIS vars föräldrar angivit att


Merparten av alla föräldrar, vare sig de rökte eller inte, visade att de var mycket angelägna att skydda sina barn mot tobaksrök. Trots att ämnet kan betraktas som känsligt idag, har svarsfrekvensen för de olika studierna varit hög och positiva kommentarer till studierna varit vanligt förekommande. Syftet med studierna har varit att bidra till ökad kunskap för att kunna ge rökande föräldrar råd om hur de bäst kan skydda sina barn. Även om dessa resultat behöver verifieras med ytterligare studier, kan resultatena redan nu bidra till att ge stöd till föräldrar om hur de bör göra för att minimera risken för att barnen exponeras för tobaksrök. Ytterligare studier behövs för att visa den kliniska betydelsen av de skillnaderna i exponeringsnivå som kunnat visas här.
ACKNOWLEDGEMENTS

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INSTÄLLNING TILL TOBAKSRÖKNING, SÄRSKILT, I NÄRHETEN AV BARN

Svara genom att markera i en ruta för varje påstående, vilken ruta beror på hur du ställer dig till respektive påstående. Vissa frågor vänder sig framför allt till rökare, men Du som ej röker har kanske en åsikt i frågan ändå.

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<tr>
<td>1. Miljöföroreningarna ute är en större fara för mitt barns hälsa än tobaksrök</td>
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<td>2. Det är mindre risk att mitt barn själv börjar röka om han/hon utsätts för tobaksrök</td>
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<td>3. Det går bra att röka i köket om man vädrar efteråt</td>
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<td>4. Jag tycker det är OK att röka bara man försöker skydda sin omgivning</td>
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<td>5. Mitt barn löper en ökad risk att drabbas av sjukdom om hon/han utsätts för tobaksrök</td>
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<td>6. Jag går alltid ut och röker oberoende av hur värdet är</td>
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<td>7. Jag skäms över att röka då jag är tillsammans med mitt barn</td>
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<td>8. Man säger bara att tobaksröken är skadlig för barn – aldrig varför</td>
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<td>9. Det är trevligare att umgås med dom som röker</td>
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<td>10. Om man verkligen var säker på att rökning är så farlig som man påstår så skulle den säkert förbjudas.</td>
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<td>13. Det är större risk att mitt barn själv börjar röka om han/hon utsätts för tobaksrök</td>
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14. Mitt barn drabbas bara av sjukdom om barnet är disponerat för denna sjukdom eller är särskilt känsligt

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15. Informationen om rökning överdrivs för att skrämmas

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16. Jag tror inte att det är bevisat att barn lättare blir sjuka om de utsätts för tobaksrök

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17. Om mitt barn blir sjukt och man kan bevisa att det beror på röken - då skulle jag sluta röka.

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18. På mödravården berättade man skräckhistorier och överdrev hur farligt det var att röka

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19. På mödravården fick jag bra stöd och hjälp med min rökning.

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20. På BVC fyller dom endast i journalen om vi röker, sen pratas det inte mer om det.

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21 BVC sköterskan har inte med att göra om vi röker.

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22. På BVC får jag ett bra stöd i samtal om rökning.

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24. Hur skulle Du vilja att personalen tar upp tobaksfrågan på mödravården?
25. Hur skulle Du vilja att personalen tar upp tobaksfrågan på barnavårdscentralen?
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

26. Om Du, som röker, skulle vilja sluta röka, eller få hjälp med att ändra rökbeteende, vilken hjälp tror Du att Du skulle ha bäst nytta av?
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

27. Mamma röker: □ □ □ □ □

28. Pappa röker: □ □ □ □ □

29. Frågorna är besvarade av □
mamma □
pappa □
mamma o pappa tillsammans □

Egna kommentarer
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________
___________________________________________________________________________

TACK för att Du svarade på våra frågor!
Frågor om rökning i hemmiljön den senaste månaden

Vår avsikt med dessa frågor är att få en så noggrann beskrivning som möjligt av hur mycket tobaksrök som kan ha funnits i hemmiljön/hemmet den senaste månaden. Försök att ange så noga som möjligt hur många cigaretter som rökt vanliga vardagar och på helgdagar. Försök sedan ange hur ofta någon rökt i de situationer som vi givit exempel på. Ange också hur viktigt det är för den som röker att göra på beskrivet sätt.

1. Hur många personer har rökt i hemmiljön den senaste månaden? _________
   (Med hemmiljö menar vi både inomhus och ute på balkong, altan, uteplats eller liknande)

2. Ungefär hur många cigaretter har sammanlagt rökt av alla rökare per dag i er hemmiljö under den senaste månaden?
   På vardagar  | På helgdagar
   0           | 0
   1–5         | 1–5
   6–10        | 6–10
   11–15       | 11–15
   16–20       | 16–20
   21–40       | 21–40
   41–60       | 41–60
   Mer än 60   | Mer än 60

3. Har någon rökt pipa, cigarr eller cigariller i hemmet? Nej □ Ja □
   Ungefär hur mycket per dag? .....................

4. Ungefär hur ofta brukar någon (även besökare) röka på följande ställen i hemmet?
   Svara med ett kryss i lämplig ruta på varje rad, tack!
   
<table>
<thead>
<tr>
<th>Flera gånger per dag</th>
<th>En gång per dag</th>
<th>Minst en gång i veckan</th>
<th>Minst en gång i månaden</th>
<th>Aldrig eller mer sällan än en gång i månaden</th>
</tr>
</thead>
<tbody>
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</tbody>
</table>
   Var som helst i inomhus  □ □ □ □ □
   Vid matbordet             □ □ □ □ □
   Vid TV:n                  □ □ □ □ □
   Vid öppen balkong/ytterdörr eller öppet fönster □ □ □ □ □
   Vid köksfläkten           □ □ □ □ □
   Ute med stängd dörr        □ □ □ □ □
   Ute med stängd dörr och klädbyte efteråt □ □ □ □ □
   Andra ställen i hemmet, nämligen: __________________________________________ □ □ □ □ □
5. Är det viktigt för någon rökare i hemmet att kunna röka på följande ställen?

*Svara med ett kryss i lämplig ruta på varje rad, tack!*

<table>
<thead>
<tr>
<th></th>
<th>Ja, absolut</th>
<th>Ja, kanske</th>
<th>Nej</th>
</tr>
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<tbody>
<tr>
<td>Var som helst i inomhus</td>
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<tr>
<td>Andra ställen i hemmet, nämligen:</td>
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</tbody>
</table>

6. Hur länge har rökvanorna i hemmet sett ut som de gör nu? 6 mån 12 mån mer än 12 mån

(dvs att det är lika många som har rökt hela tiden, att de har rökt lika mycket som nu och på samma ställen i hemmiljön som nu)

7. Hur ofta är Ditt barn i miljöer (utanför hemmet) där det förekommer tobaksrökt? Aldrig Sällan Varje vecka Varje dag

Om ja, var? ______________________________

Egna kommentarer: ______________________________________________________________
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________

**Hjärtligt Tack för hjälpen!**