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Exposure to Environmental Tobacco Smoke in Babies

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1. Introduction

1.1 What is passive smoking?

Passive smoking is defined as the involuntary inhalation of tobacco smoke in closed spaces. It is also known as second hand smoke (SHS) or environmental tobacco smoke (ETS) (ENHS, 2009). ETS includes:

- *Mainstream*: Smoke exhaled by smokers
- *Sidestream*: Smoke produced during the combustion of cigarettes. Approximately 70% of ETS is made by side stream. This smoke is generated by components that escape through every pore of the filter and the paper of the cigarette. The sidestream contains small gaseous particles that can reach peripheral areas of the lung. The sidestream shows 3 more times tar and nicotine than the smoke directly inhaled by the smoker, and 5 more times carbon monoxide (CO) (US Surgeon General, 2006).

In each cigarette, 4000 chemical agents have been found, 43 of them highly toxic such as arsenic, ammonia, benzene, cadmium, hydrogen cyanide, polonium 210, butane, CO, cyanide and acetone (Us Surgeon General, 1989) (see Appendix 1).

Scientific research has demonstrated that pollution caused by tobacco smoke at home can generate higher contamination rates than contamination by industrial air pollution. A series of publications have shown that children are exposed to ETS by 59.4 % - 75 % because of maternal smoking. Up to 70 % of the western children live in houses where at least one of the parents smokes. Thirty percent of these children are exposed to tobacco smoke every day (King et al., 2009).

Passive smoking is the leading cause of preventable death in infancy in industrialized countries (third cause of preventable death in adults), and one of the first leading causes of cancer in developing countries (Seong et al., 2010; Royal College of Physicians, 2010). Some authors estimate that, in Spain, between 1,228-3,237 deaths of lung cancer and cardiovascular diseases are attributable to ETS exposure at home and at work. This circumstances clearly show that exposure to passive smoking is an important public health issue (Lopez et al., 2002).

In 1950 Doll and Hill were the first to publish scientific work related to the harmful health effects of tobacco consumption (Doll & Hill, 1950). In 1974 medical research began to headline harmful effects of ETS on children's health caused by parental smoking (Harlap & Davies, 1974). Furthermore, during the last two decades, international research has found that children of smokers are more likely to be adolescent smokers, probably as a result of role models or easy access to cigarettes (Roseby et al., 2003). Nowadays numerous harmful effects of ETS have been associated with child health (Us Surgeon General, 2006). According to the International Agency for the Research on Cancer (IARC) of the World Health Organization (WHO), ETS is a type A carcinogen and no safety threshold has been established yet (IARC, 2002). Pediatric illnesses and disease related to environmental tobacco smoke are: increased risk of sudden infant death syndrome (SIDS), acute respiratory diseases, respiratory symptoms, aggravation of symptoms in patients with asthma, acute and chronic diseases in the middle ear and slowed lung growth (IARC, 2020; Samet & Sockrider, 2011).

2. Effects of passive smoking in children

2.1 Sudden Infant Death Syndrome (SIDS)

Sudden infant death syndrome (SIDS) can be defined as the sudden and not expected and unexplained death of an infant younger than one year, after a meticulous investigation of potential factors that might have lead to infant death –including an autopsy, the examination of the death scene and the revision of the clinical history (Hunt, 2001).

According to Hannah and collaborators (Kinney & Thach, 2009) there exists no consensus about how SIDS should be defined. For instance, the definition of SIDS found in Medline reads as follows: the sudden, unexplained death of an infant younger than one year old. Some people call SIDS "crib death" because many babies who die of SIDS are found in their cribs (MedlinePlus, 2011). The National Institute of Health consensus conference issued the first standardized definition of sudden infant death in 1969 and defined it as the ‘sudden death of an infant or young child, which is unexpected by history, and in which a thorough post mortem examination fails to demonstrate an adequate cause of death. The definition required thus an autopsy for all infants, who died from a similar condition as SIDS to establish a set of infants showing similar characteristics for whom vital statistics, research,

and family counseling were needed' (Hannah et al., 2009). Furthermore, the authors stated that 'although SIDS was defined as a syndrome, and thus potentially the result of more than one disease, many observers still viewed SIDS as a single entity, because of its distinctive features, which included a peak incidence at 2 to 4 months of age, male predominance, and the presence of intrathoracic petechiae. Subsequent modifications of the definitions of SIDS restricted the application of the diagnosis, SIDS, to infants under the age of 12 months, added the requirement of a death-scene investigation, or linked the death to a sleep period (i.e., the time when the majority of deaths occurred). It is still unclear whether SIDS occurs during sleep or during the many transitions between sleep and arousal that occur during the night, since such deaths are typically not witnessed'. It has to be noted that heterogeneity of the definitions of SIDS has lead to contradictory results in scientific studies (Kinney & Thach, 2009).

The incidence of SIDS in industrialized countries ranges between the lower taxes found in Japan (0.09 cases per 1000 infants) to the higher taxes found in New Zealand (0.80cases per 1000 infants); United States has an intermediate rate (0.57 cases per 1000 infants) (Moon et al. 2007).

Epidemiological studies have identified several risk factors for SIDS, which are mainly related to pregnancy and postnatal period. Among these risk factors, mother's tobacco consumption represents one of the most important preventable causes of SIDS. For instance, if one third of the mothers smoked during and after pregnancy, 25 % of all cases of SIDS might be attributable to this (Carrion & Pellicer, 2002).

A quantitative systematic revision has found that infant, who suffered SIDS were two times more exposed to passive smoking than those who were not (OR=2.08 and 1.94 for prenatal and postnatal maternal tobacco consumption, respectively). There is evidence showing that there is a higher postnatal exposition to ETS, when both parents smoke during pregnancy (Anderson & Cook, 1997).

A recent ecological study conducted from 1995 to 2006 has shown that for every 1% absolute increase in the prevalence of smoke-free households with children, the rates of SIDS decrease by 0.4% (Behm et al., 2011.

2.2 Respiratory symptoms and illness

Several studies have shown an increased frequency of common respiratory symptoms of children (cough, sputums and wheezes) when parental smoking was present (US Surgeon General, 2006; Cook & Strachan, 1999) . A meta-analysis conducted by Samet and Sockrider revealed that parental smoking was associated with respiratory symptoms (asthma, wheeze, cough, rise of sputums or dyspnea of their children (OR ranged between 1.23 and 1.35). The highest risks were found when both parents smoked (see Table 1) (US Surgeon General, 2006).

2.2.1 Infections of the lower respiratory tract

During the first years of life of children, increases in respiratory problems of children are related to parental smoking habits. If both parents smoke the presence of wheezes and asthma increases by 20%. At the same time, infections of the lower respiratory tract and

cough increases by 30% and by 13%, respectively (Zmirou et al. , 1990). These circumstances are directly related to the consumption of tobacco by the mother. For instance, bronchial infection with wheezes are 14% more frequent when mothers smoke more than 4 cigarettes per day, and 49% more frequent when they smoke more than 14 cigarettes per day (Neuspiel et al., 1989).

	Either parent smokes			One parent smokes			Both parents smoke		
	N	OR	95%CI	N	OR	95%CI	N	OR	95%CI
Asthma	31	1.23	(1.14-1.33)	7	1.01	(0.84-1.22)	10	1.42	(1.30-1.56)
Wheeze *	45	1.26	(1.20-1.33)	13	1.18	(1.10-1.26)	14	1.41	(1.23-1.63)
Cough	39	1.35	(1.27-1.43)	18	1.27	(1.14-1.41)	18	1.64	(1.48-1.81)
Phlegm Δ	10	1.35	(1.30-1.41)	7	1.24	(1.10-1.39)	6	1.42	(1.19-1.70)
Breathlessness Δ	6	1.31	(1.14-1.50)						

N: Number of studies

* Excluding EC study, in which the pooled odds ratio (OR) was 1.20

Δ Data for phlegm and breathlessness restricted as several comparisons are based on fewer than five studies.

Adapted from USDHHS 2006, The Surgeon General's Report and Jonathan M Samet, Marianna Sockrider, Secondhand smoke exposure: Effects in children (Available at: www.uptodate.com, last accessed 19th October 2011)

Table 1. Effects for respiratory symptoms in children whose parents smoke according to the meta-analysis conducted by Samet and Sockrider

Bronchiolitis usually takes place among the first six months of life. Children with bronchiolitis history tend to present more bronchial spasms during early years and asthma among adolescence. This is why bronchiolitis is often considered as a risk factor that precedes repeated crisis of bronchial spasms and wheezes until the age of eight. Subsequently, passive smoking constitutes a risk factor for the appearance for asthma symptoms in infancy (Martin et al., 2003). Moreover, several studies have found that after the implementation of more restrictive tobacco law, hospitalizations of infants due to asthma decreased by 18.2% (Carriazo & Cuevo, 2010; Mackay et al., 2010).

Recently, a systematic revision including a meta-analysis has been published. The objective of this study was to explore if family tobacco consumption is related to low respiratory tract infections (LRTI) in children. This meta-analysis included more than 60 studies and showed that, if the father smokes, the OR for LRTI is 1.22 (95%CI: 1.10-1.35), if both parents smoke, the OR for LRTI is 1.62 (95%CI: 1.38-1.89) and that if any other member of the family also smokes, the OR for LRTI rises up to 1.62 (95%CI: 1.45-1.73). The strongest association of passive smoking with bronchiolitis was found when any other member of the family, besides the parents, also smoked (OR: 2.51; 95%CI: 1.96-3.21). Consequently, the authors concluded that passive smoking of cohabitants in children's households is the most important risk factor for LRTI. This risk is especially high, if the mother smokes during the postnatal period (Jones et al., 2011).

In some studies LRTI has been related to maternal tobacco consumption, but several oriental countries, as for example China and Vietnam, where maternal tobacco consumption is not

frequent, show that paternal tobacco consumption itself can explain the rise of the incidence of LRTI (Jones et al., 2011; Suzuki et al., 2009; Chen et al., 1986).

Certain studies have shown that the effect of passive smoking of babies on respiratory illnesses is critical during the first year of life, since this is the period when the baby stays more time with his or her parents, even though important effects of passive smoking on respiratory illnesses have been found during school age (Oberg et al., 2011).

2.2.2 Asthma

The etiology of asthma is yet not clearly established. Some authors have related asthma to a higher frequency of respiratory infections in early life or to other inflammatory mechanisms of the pulmonary epithelium (Boulay & Boulet, 2003).

Several published scientific revisions have shown that there is a clear relationship between the prevalence and severity of asthma and the exposition to tobacco smoke in early life (US Surgeon General, 2006; Cook & Strachan, 1999).

Some authors speculate that smoking during pregnancy can alter pulmonary development in utero, which can be related to a higher risk for the development of asthma later in life (Milner et al., 1999). Other authors consider that SHS might be closely related to an allergic sensitization, however not a clear a clear association between tobacco smoke exposure and allergy has been established (Cook & Strachan, 1999).

There is clear epidemiologic evidence for an association of exposition to SHS with the risk to suffer asthma in childhood (US Surgeon General, 2006; Goksor et al., 2007). Table 1 shows the excess risk when parents or only the mother smokes. Although there is a dose response relationship between SHS and asthma, a well defined safety threshold level without risk has not been established (Institute of Medicine, 2007). Surgeon General's Report in 2006 revealed a relationship between the prevalence of SHS and asthma, but not with the incidence of asthma (US Surgeon General, 2006). Moreover, the exposure to tobacco smoke during early years is associated with a higher prevalence of asthma in adults (Larsson et al., 2001; Svanes et al., 2004).

Finally, numerous studies have related the tobacco smoke exposure to different clinical outcomes such as an increase in a) number of visits to the emergency departments due to asthma, b) clinical symptoms, c) number of medicines and d) other clinical parameters (Carlsen & Lodrup, 2005).

2.2.3 Other respiratory illnesses

Altet and collaborators found a positive association between passive smoking in infancy and a higher risk to develop pulmonary tuberculosis (Altet et al., 1996).

2.3 Middle ear illnesses

Some studies have suggested an association of parental smoking with the presence of middle ear illnesses in children. (Adair-Bischoff & Sauve, 1998; Kum-Nji et al. 2006). The meta-analysis conducted by the US Center of Disease Control and Prevention found a

pooled odds ratio of 1.37 (95% CI 1.10-1.70) for recurrent middle ear otitis, 1.33 (95% CI 1.12-1.58) for middle ear effusions, and 1.20 (95% CI 0.90-1.60) for clinical referrals or operative interventions for middle ear effusions, if either parent smoked (US surgeon General, 2006).

Another meta-analysis concluded that middle ear illnesses were associated to prenatal smoking (OR:1.1), postnatal smoking (OR:1.46) and paternal smoking (OR: 1.27), the association is even higher if cohabitants, apart from the parents, smoke at home (OR: 1.35) (Royal College of Physicians, 2010).

2.4 Meningitis

A recently published article shows a significant positive association between passive smoking with invasive meningococcal bacteriological illness, as well as an increased carriage of *N. meningitides* and *S. pneuomoniae* (Lee et al., 2010). Another recent scientific revision revealed that parental smoking (one or both parents) doubled child's risk to suffer a meningitis (OR: 2.30; 95%CI: 1.74-3.06) (Tobacco Advisory Group, 2010).

2.5 Dental caries

A possible effect of second hand smoke on the development of dental caries in children is not clearly established; a recently published scientific revision has failed to demonstrate a potential association of second hand smoke with caries, maybe due to the heterogeneity of the studies included (Hanioka et al., 2011).

3. Strategies to reduce passive smoking in babies

There are four main strategies to try to reduce exposure to passive smoking of babies: education, regulation, legislation and litigation (Davis, 1998). These strategies mainly depend on administrative measures. Nevertheless, the role of health professionals concerning the education of their patients is critical, in order to protect non-smokers from passive smoking and promote smoke-free spaces (Samet & Sockrider, 2011).

The role of pediatricians and especially pediatrics residents is of vital importance as concluded in a study published in 2008 (Hymowitz et al., 2008). The study evaluated the efficacy of a special program for training pediatric residents to address tobacco and reduce exposure to tobacco smoke. As stated by authors 'The percent of parents who smoke at sites associated with the special training condition, but not of those at sites associated with standard training, who reported that residents advised them to stop smoking, offered to help them quit, and provided quit smoking materials increased significantly from baseline to year 4'.

The above listed strategies should also focus on women in reproductive age and on pregnant women, since both conditions are the two first exposure patterns of exposure to tobacco among babies. Different types of interventions have been developed and applied to promote smoking quitting during pregnancy and in the maternal-child environment (Jane et al., 2006; Salleras et al., 2003).

The emergence of health protection laws has addressed limitations of tobacco use in public. Closed spaces for smokers have been proved to be useful measures to prevent respiratory

diseases in workers. For instance, the Spanish law 42/2010 prohibits the exposure of babies to tobacco smoke in public, closed places. However it does not regulate the exposure in private areas such as in the car or at home. Hence, a pediatric program in Primary Care should be defined and promoted, including interventions among families to reduce the exposure to SHS in children. In Spain pediatricians regularly follow up children's health status, and they would be the most appropriate professional group to promote passive smoke protection among children (Altet et al., 2007; Ortega et al., 2010).

In order to reduce infant exposure to ETS in private spaces, it is necessary to encourage parental (or baby sitters) smoking cessation. If not possible, smoke-free households could be promoted as well as the avoidance of smoking in the presence of a baby.

In Spain, the above mentioned law increases the awareness of smokers about the convenience of smoking in open-air spaces to protect others at work and public places; consequently parents, who smoke are more predisposed to adopt strategies to protect their children and other family members (see Appendix 2).

According to the last national survey of health (2006), the prevalence of adult smokers in Spain is 26.44% (21.51% in women and 31.56 in men) (see Appendix 3); in young adults the prevalence ascends up to 35 %, in spite of the fact that in the last years the number of smokers generally has decreased. Nonetheless, the number of babies exposed to tobacco smoke is still considerable (INE, 2006). Consequently, the reduction of the prevalence of smoking individuals (mainly the parents or the baby sitters) continues to be the most effective way to reduce exposure to passive smoking among infants. The following policies have contributed substantially in the reduction of the prevalence of smokers: a) increment of policies to control tobacco consumption, b) increased tobacco taxes, c) promotion of awareness about tobacco related health problems by informative advertisement, campaigns, and d) the increase of sanitary protection devices to help smokers in their cessation (Tobacco advisory Group, 2010). Programs directed to reduce the prevalence of smokers are those measures that mainly contribute to achieve smoke-free domiciles (Thomson et al., 2006).

4. Theories of change

In the previous chapter, we review the strategies to reduce ETS exposure in children. However, ultimately, reduce the exposure of children depends on parents and / or caregivers. Therefore, in this section, we review the theoretical foundations and implementation, based on some articles that determines the effectiveness of interventions to reduce children's exposure to passive smoking, and how has been carried out by parents. It is a systematic review of 2008 (Priest, N.), which included 36 studies. Sixteen of 36 them expressly employed a theoretical framework in the design and/or development of the intervention.

In 1994 McIntosh developed the activities for the parent manual based on behavior modification theory. Groner 2000 employed the health belief model, and Wakefield 2002 used a harm minimization approach. Abdullah 2005 based counseling strategies on the stages of change component of Prochaska's transtheorical model. Krieger 2005 was also guided by the trastheorical stages of change model, as well as by social cognitive theory.

The Motivational Interviewing, was used by Emmons 2001, Curry 2003 and Chan 2005. Chan 2006a used Ajzen's theory of planned behavior in the development of their educational intervention.

Greenberg 1994, Elder 1996, Conway 2004 and Fossum 2004 employed the social learning model.

Now, in this section we will review the most important theories concerning modifications of behavioral patterns will be presented. We have divided them in three levels following the ecological perspective of the scientific revision made by Glanz and collaborators in 1997 (Glanz et al., 1997).

CONCEPT	DEFINITION
Intrapersonal level	Individual characteristics that influence behavior, such as knowledge, attitudes, beliefs, and personality traits
Interpersonal level	Interpersonal processes and primary groups, including family, friends, and peers that provide social identity, support, and role definition
Community level	
Institutional factors	Rules, regulations, policies, and informal structures, which may constrain or promote recommended behaviors
Community factors	Social networks and norms, or standards, which exist as formal or informal among individuals, groups, and organizations
Public policy	Local, state and federal policies and laws that regulate or support healthy actions and practices for disease prevention, early detection, control and management

Note: Table published by Glanz et al. 1997

Table 2. An ecological perspective: Levels of influence

4.1 Individual or Intrapersonal level

The Health belief model by Janz and Becker (Janz & Becker, 1984) proposes that when someone deems it best to change a behavioral pattern, both advantages and disadvantages of this change are taken in consideration, followed by a rational decision. In addition, the perception of the individual susceptibility to the problem that requires a behavioral change and the severity of the problem are both taken into account. Recently, the concept of auto-efficiency has been added to this model. Auto-efficiency, a concept originally taken from Bandura's work (Bandura, 2011), defines an individual's confidence in undertaking a specific change in conduct.

The Health action model by Tones (Tones, 1991) is based on the previous model, but incorporates also the concept of self-esteem. Self-esteem is defined in this model as the individual's beliefs on his or hers qualities, and how they are perceived by others. Furthermore, this model includes a number of factors, such as previous knowledge and environmental features that can influence decision-making in the process of individual behavioral change.

The Theory of Reasoned Action / Planned Behavior by Ajzen (Ajzen, 1991) characterizes conduct depending on behavioral intention, subjective regulations and attitudes. This model specifies that real behavior is predicted by someone's intention to realize a

behavior. Hence, intention is the result of attitudes and subjective rules. Ajzen later modified and included the concept of "perceived behavioral control" that reflects, how easy or difficult an individual can perceive the task to follow a certain behavioral pattern.

The Transtheoretical Model was proposed by Prochaska y DiClemente (Prochaska & DiClemente, 1983). This model describes the phases in conduct change, and is one of the newest models describing behavioral change. It is also known as 'transtheoretical model' since it incorporates the structures of other, older models. This model considers behavioral change as a dynamic process –instead of a static process-, which takes into account the fact that, rapidity for behavior change is different in each individual and that changes occur in time following five stages. This stages are: 1) precontemplative: the individual does not think about possible behavioral changes; 2) contemplative: the individual is thinking about a change; 3) preparation: the individual enters a necessary path to be able to perform a conduct change; 4) action: the individual carries out the change in a short period of time; and 5) maintenance: the individual maintains a behavioral change over a period of time, (usually measured as keeping the change for at least six months). The transtheoretical model includes the possibility of relapses in earlier phases. This means that a maintained behavior change can also be achieved after a cyclical process of progresses and relapses. This model has been broadly used, but continues to be controversially discussed (Katz, 2001; Littlell et al., 2002; van Sluijs et al., 2004; Whitelaw et al., 2000).

The Precaution Adoption Process Model proposed by Weinstein (Weinstein, 1988), This model includes seven phases: not being informed or aware of a problem (unprepared), without interest in the problem, deciding about intervention, determined not to act, acting and maintenance. These phases represent qualitative models of different behavioral patterns, beliefs and experiences, and the factors influencing the transitions between the different phases can vary. Weinstein believes that this model is linear, and that interventions require being adapted to each phase of the model. It displays similarities to the transtherorical model of behavioral change.

4.2 Interpersonal level

Social learning theory or social cognitive theory of Bandura (Bandura, 2001) postulates that people learn by observing someone else's behavior and apply it to themselves. Both, the credibility and reinforcement of the learned conduct are fundamental. This theory goes beyond individual factors influencing behavioral changes. It includes environmental and social factors. The key elements of this theory are summarize in table 3 (Cooper, 2000; Redding et al., 2000). The self-efficacy has been evaluated as an important predictor of the success in the proposed behavioral change (Cheng, 1999).

Differential Association-Reinforcement Theory (Glanz et al., 1997) is based on the circumstance that we can adopt new behavioral patterns by looking and imitating other persons. There are positive and negative reinforcements (price and punishment, for example). It mainly takes into account what happened after conduct change. Behavioral patterns are imitated leading to different levels of reinforcement, taking into account exposure and consequences of the conduct.

Theory	Focus	Key Concepts
Health Belief Model	Considers the pros and cons for behaviour change, as well as the perception, susceptibility and severity of the illnesses	Perceived susceptibility Perceived severity Perception of benefits from action Action barriers Auto-efficiency
Health Action Model	To promote people's self-esteem. To find environment support	Self-esteem Own beliefs Knowledgements Favourable environment
Transtheoretical Model	The intention to change varies among individuals and in time periods. The relapse is a normal phase in the process of change	Precontemplation Contemplation Preparation Action Manteinance Relapse
The Precaution Adoption Process Model	Changes need of certain phases and intervention should be adapted to those phases.	Without information Without interest Deciding what to do Decided to act Decided not to act Acting Manteinance
Social Learning Theory or Social Cognitive Theory	Considers the credibility of social models. Positive reinforcement. Helps conductal maintenance	Environment Situation Conductal capacity Expectation Hope Self-control Observational learning Reinforcements Self-efficacy Emotional aspects Reciprocal determinism
Differential Association-Reinforcement Theory	We learn new behavioural patterns by looking at and imitating others and by positive and negative reinforcements. The subject defines a behavioural pattern as good or justified according to his or her commitment. It takes into account what happens once a conduct change initiated.	Imitation Positive reinforcements Negative reinforcements Consequences of the behaviour
Self-Regulation Theory	Behaviour is defined by the interaction between subject and environment. Self-efficacy is a key concept as well as the personal standards.	Interaction environment-subject Self-efficacy Personal standards
Harm reduction model	Necessity to reduce health risks related to drug consumption, at both individual and community level.	Respect Acceptance Support Promotion of own's capacities (empowerment)

Table 3. Summary of the theories of change

4.3 Community level

Self-Regulation Theory (Baumeister et al., 2004). Behavior is seen as the sum of interactions between the individual and environment. Self-efficacy is the key question. Response is related to our personal values. It has been studied in the following fields: exercise, stress, alimentary behavior, cardiovascular disease and drug consumption.

Harm reduction model developed, among others, by Newcombe and O'Hare (Newcombe et al., 1995) is based on the principles of respect, acceptance, support and promotion of the own capacities (empowerment). This model was generated as an answer to the accumulating evidence of serious health risks related to alcohol overconsumption and tobacco use. Later on, it has been used for the understanding of behavioral pattern changes related to the consumption of drugs and to HIV infection. The model aims to diminish the negative effects of drug consumption, and does not imply quitting completely the drug consumption (O'Hare et al., 1995).

Some authors (Nigg et al., 2002), postulate that these theories can help us to elucidate, why a person is motivated to initiate a behavioral change. Other authors, in contrast, emphasize more on the reasons, why a certain conduct is being hold in time. They also suggest that for different health problems, different models can be useful, which at different stages of behavioral change can explain better these changes. Finally, multiple interventions concerning a series of health related problems (for instance, obesity and tobacco use or alcohol consumption) may need inputs from several theories at different phases or stages, to be able to explain in an improved way behavioral patterns (Martin, 2006).

5. Conclusions

There is enough scientific evidence to conclude that indirect tobacco smoke exposure of children (and adults) has negative health effects. Tobacco smoke exposure of children can cause: low tract respiratory infections, middle ear infections, asthma, low weight at birth and sudden death syndrome.

Subsequently, strategies to reduce passive exposure to tobacco are an urgent and priority sanitary, public health aim. Scientific evidence should enforce actions performed by health care professionals of all disciplines to undertake measures in order to eradicate this avoidable risk in children.

6. Key points

- Environmental tobacco smoke is made from the smoke exhaled by smokers and the smoke produced during the combustion of cigarettes.
- Passive smoking is the leading cause of preventable death in infancy in industrialized countries and one of the first leading causes of the rise of cancer in developing countries.
- Up to 70 % of the western children live in households where at least one of the parents smokes. Thirty percent of these children are exposed every day to tobacco smoke.
- Sudden infant death syndrome, acute respiratory illnesses, acute and chronic middle ear infections and meningitis are related to ETS.

- Passive smoking during pregnancy lowers baby's weight at birth, highers the risk of: prematurity, congenital malformations, spontaneous abortion and fetal and perinatal mortality.
- The rise of the policies controlling tobacco consumption, the increase in of tobacco taxes, the awareness of tobacco related health problem in advertising informative campaigns, and the increase of sanitary devices to help smokers in their cessation have contributed to reduce the number of smokers.
- Reducing the prevalence of smokers among parents and baby sitters continues to be the most effective way to prevent passive smoking among children.

Compound	Does it cause cancer?
Nicotine	
Dimethylnitrosamine	
Benzopyrene	X
Pyrene	X
Napthalene	X
Phenol	
Acetone	X
Methanol	
Vinyl	
Carbon Monoxide	X
Nitrogen oxides gases	
Tolue	
Benzene	
Arsenic	
Cadmium	X
Niquel	
Lead	
Naphthylamine	X
Toluidine	X
Polonium-210	X
Ammonia	
Dibenzocridine	X
Cianamide	
DDT	
Uretane	
Butane	

Adapted from: Departament de Salut de la Generalitat de Catalunya (http://www.gencat.cat/salut/depsalut/pdf/gtabacp.pdf, last accessed November 10th 2011) and Cancer Society of New Zeland Inc. and Health Promotion Services Branch Health Dept. of Western Australia

Appendix 1. Main components of tobacco smoke and its carcinogenic effect

Exposure	Does NOT avoid exposure	Avoids exposure
At home	To smoke at home when the bay is not there	Not smoking at home, in any room
	To smoke in delimitated areas of the house	Prohibit family members and visitators to smoke in the house
	To smoke with the window or balcony open	To always smoke outside the house. To smoke only in the balcony or terrace keeping the door closed
	To smoke and ventilate the house after smoking	
In the car	To smoke in the car when the bay is not there	Not to smoke in the car, even though the baby is not there
	To smoke in the car when the baby is there but with the window opened	

From: Ortega G, Castellà C, Martín-Cantera C, Ballvé JL, Díaz E, Saez M, et al. Passive smoking in babies: the BIBE study (Brief Intervention in babies. Effectiveness). BMC Public Health. 2010;10:772.

Appendix 2. Measures to avoid exposure to tobacco smoke in babies

	Number	%
Men		
Total	5,794.60	31.56
16-24 years old	596.3	24.96
25-34 years old	1,580.90	40.16
35-44 years old	1,382.40	37.41
45-54 years old	1,141.30	38.83
55-64 years old	654.2	28.45
65-74 years old	312.4	18.64
≥75 years old	127	8.93
Women		
Total	4,101.70	21.51
16-24 years old	657	28.93
25-34 years old	1,108.40	30.16
35-44 years old	1,093.00	30.73
45-54 years old	845.6	28.47
55-64 years old	286.5	11.77
65-74 years old	83.9	3.86
≥75 years old	27.3	1.37

From: Spanish Ministry of Health. National Health survey, 2006

Appendix 3. Tobacco consumption in Spain. Daily number of smokers (in thousands) and percentage aged ≥ 16 according to sex and age group

7. Abbreviations

CI: Confidence Interval
CO: Carbon monoxide
ETS: Environmental Tobacco Smoke
IARC: International Agency for the Research on Cancer
LRTI: Low respiratory tract infections
OR: Odds Ratio
SHS: Second Hand Smoke
SIDS: Sudden Infant Death Syndrome
WHO: World Health Organization

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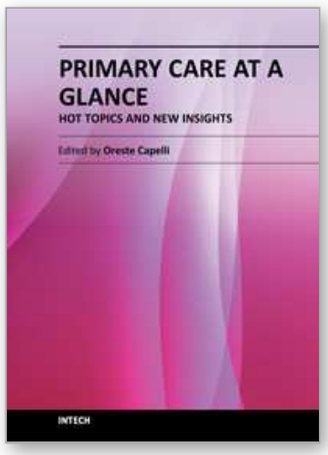
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