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# Tabaquismo pasivo infantil: otras patologías y riesgos asociados


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# Objetivos docentes

- Al final de la presentación, el asistente será capaz sostener con pruebas que el tabaquismo pasivo en niños:
  - Aumenta ***la caries dental***
  - Se asocia a menor ***desarrollo neuromotor***
  - Aumenta el riesgo de ***obesidad y riesgos cardiovasculares***
  - Se asocia a ***disfunción cardiovascular***
  - Parece asociarse a ***dermatitis atópica***
  - ***Es caro***
  - En general, ***es malo para todo***.

# Introducción

- “El tabaquismo pasivo, de segunda y tercera mano, es perjudicial para cualquier aspecto de la ***salud*** de los niños y de la ***economía*** de los padres y la sociedad en la que se desarrolla.
  - Es malo
  - Sale caro
  - Huele.”

# Tabaquismo pasivo infantil

- Tabaquismo pasivo infantil
  - Patología respiratoria
  - Patología oncológica
  - ***Otras patologías y riesgos asociados***

# Las grandes revisiones: tabaquismo activo

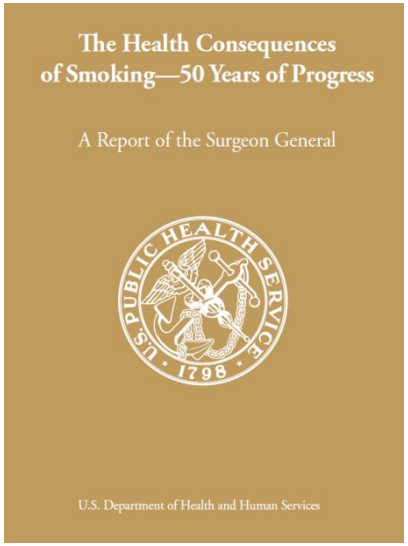
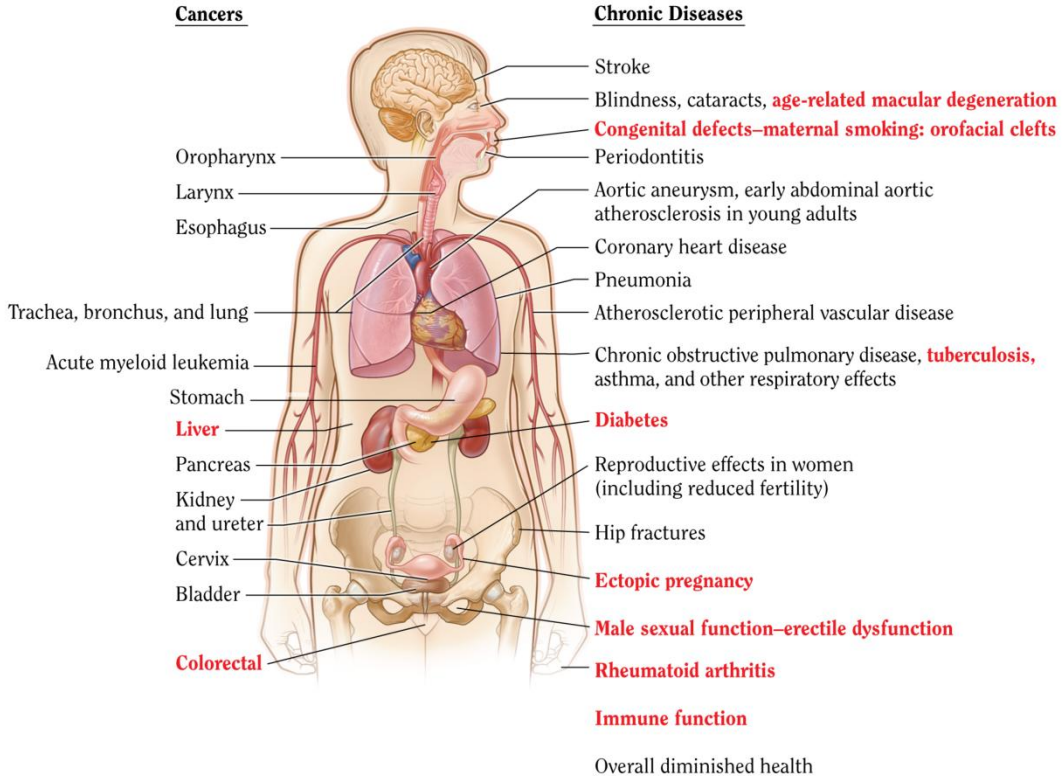
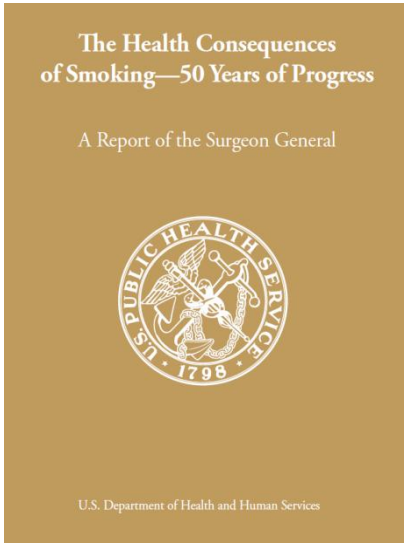



**Figure 1.1A The health consequences causally linked to smoking**



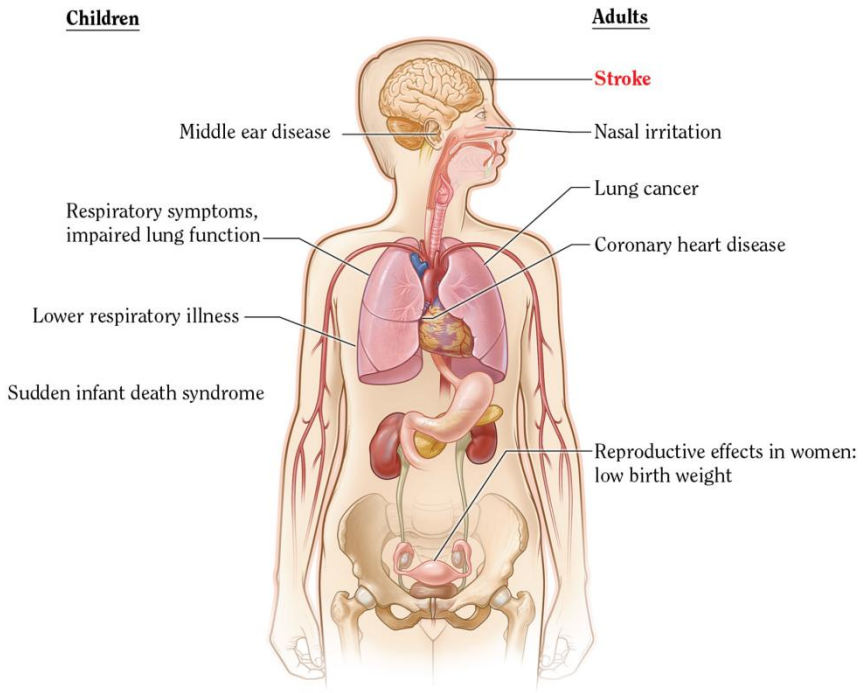

*Source:* USDHHS 2004, 2006, 2012.
*Note:* The condition in **red** is a new disease that has been causally linked to smoking in this report.

# Las grandes revisiones: tabaquismo pasivo




*The Health Consequences of Smoking—50 Years of Progress*

**Figure 1.1B The health consequences causally linked to exposure to secondhand smoke**




*Source:* USDHHS 2004, 2006.
*Note:* The condition in **red** is a new disease that has been causally linked to smoking in this report.

# Tabaquismo pasivo infantil: ¿hay más?

THE JOURNAL OF PEDIATRICS • www.jpeds.com ORIGINAL ARTICLES

Secondhand Tobacco Smoke Exposure and Neuromotor Function in Rural Children

Samrat Yeramaneni, MBBS, PhD[1,2], Kim N. Dietrich, PhD[1], Kimberly Yolton, PhD[3], Patrick J. Parsons, PhD[4,5], Kenneth M. Aldous, PhD[4,5], and Erin N. Haynes, DrPH[1]

**Objective** To investigate the relationship between secondhand tobacco smoke (SHS) exposure and neuromotor function in children.

**Study design** We studied 404 children aged 7-9 years who were exposed to SHS and other environmental neurotoxicants. Parents reported smoking habits, and serum cotinine levels were measured in children to determine SHS exposure. The Halstead-Reitan Finger Oscillation Test, Purdue Grooved Pegboard Test–Kiddie version, and Bruininks-Oseretsky Test of Motor Proficiency 2–Short Form were used to assess neuromotor function. Multivariable regression models that accounted for potential confounders were used to evaluate the associations.

**Results** Approximately 50% of the children were exposed to SHS based on serum cotinine measures. Exposure to SHS was significantly associated with motor impairment in children, including diminished visuomotor coordination ($P = .01$), fine motor integration ($P = .01$), balance ($P = .02$), and strength ($P = .04$) after adjusting for exposures to lead and manganese, age, sex, body mass index, measures of parental cognitive abilities, parental education, and quality of home environment.

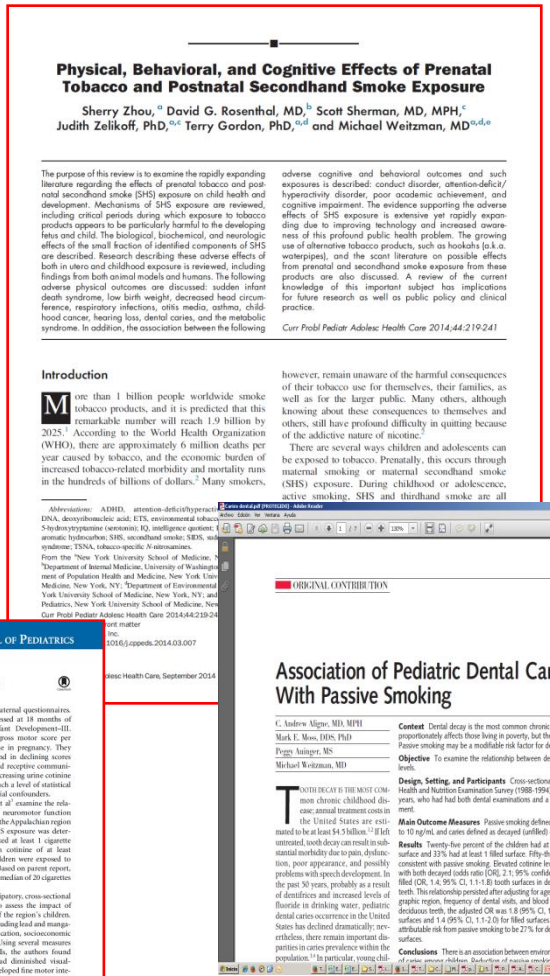
**Conclusion** SHS is a neurotoxicant that may be associated with impaired childhood neuromotor function. *(J Pediatr 2015;167:253-9).*

See editorial, p 224 and related article, p 246

There is compelling evidence from several studies supporting the association between secondhand tobacco smoke (SHS) exposure and increased risk of learning disabilities,[1,2] attention deficit disorder/attention deficit hyperactivity disorders,[3,4] behavior and conduct disorders,[5-7,10] and cognitive and academic achievement deficits in children.[11-14] There is little research, however, on the effects of SHS exposure on neuromotor function, an important component of neuropsychological function, which is related to overall cognitive attainment and achievement in clinical pediatric populations[15] as well as in typically developing children.[16,17] Indeed, there is strong evidence from research on children with developmental disorders and emerging evidence from typically developing children indicating that motor skills and cognitive abilities are interrelated at the behavioral level[15,18-20] and the neuroanatomic level.[16,17,21]

The few published studies examining the role of prenatal tobacco smoke exposure on motor development in children at different ages have reported equivocal results. Trasti et al[22] found a small but significant negative association between maternal smoking during pregnancy and motor development in children at age 5 years. Barr et al[23] reported a significant relationship between prenatal exposure to tobacco and lower examiner ratings on fine and gross motor skills in children at age 4 years; however, this relationship did not persist after adjusting for significant outliers (subjects reporting >3.5 cigarettes/day), and other covariates, including prenatal education, prenatal nutrition, ma... by Richardson et al[24] and Fried ... associations between prenatal ex... development in children. Herna... (48-72 hours old) of mothers w... to SHS based on self-reported ... performance, quality of moveme...

| | |
|---|---|
| BMn | Blood manganese |
| BOT-2 | Bruininks-Oseretsky Test of M... |
| BPb | Blood lead |
| HMn | Hair manganese |
| HRFOT | Halstead-Reitan Finger Oscill... |
| PGPT | Purdue Grooved Pegboard T... |
| SHS | Secondhand tobacco smoke |



**Physical, Behavioral, and Cognitive Effects of Prenatal Tobacco and Postnatal Secondhand Smoke Exposure**

Sherry Zhou,[a] David G. Rosenthal, MD,[b] Scott Sherman, MD, MPH,[c] Judith Zelikoff, PhD,[o,c] Terry Gordon, PhD,[o,d] and Michael Weitzman, MD[o,d,e]

The purpose of this review is to examine the rapidly expanding literature regarding the effects of prenatal tobacco and postnatal secondhand smoke (SHS) exposure on child health and development. Mechanisms of SHS exposure are reviewed, including critical periods during which exposure to tobacco products appears to be particularly harmful to the developing fetus and child. The biological, biochemical, and neurologic effects of the small fraction of identified components of SHS are described. Research describing these adverse effects of both in utero and childhood exposure is reviewed, including findings from both animal models and humans. The following adverse physical outcomes are discussed: sudden infant death syndrome, low birth weight, decreased head circumference, respiratory infections, otitis media, asthma, childhood cancer, hearing loss, dental caries, and the metabolic syndrome. In addition, the association between the following adverse cognitive and behavioral outcomes and such exposures is described: conduct disorder, attention-deficit/hyperactivity disorder, poor academic achievement, and cognitive impairment. The evidence supporting the adverse effects of SHS exposure is extensive yet rapidly expanding due to improving technology and increased awareness of this profound public health problem. The growing use of alternative tobacco products, such as hookahs (a.k.a. waterpipes), and the scant literature on possible effects from prenatal and secondhand smoke exposure from these products are also discussed. A review of the current knowledge of this important subject has implications for future research as well as public policy and clinical practice.

*Curr Probl Pediatr Adolesc Health Care 2014;44:219-241*

**Introduction**

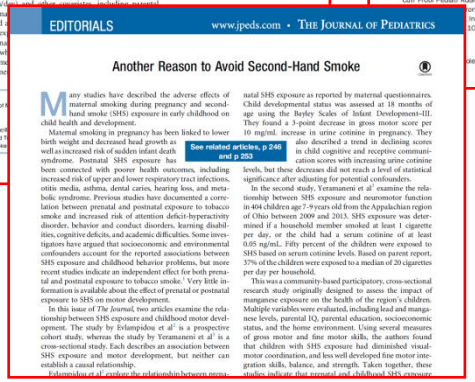
More than 1 billion people worldwide smoke tobacco products, and it is predicted that this remarkable number will reach 1.9 billion by 2025.[1] According to the World Health Organization (WHO), there are approximately 6 million deaths per year caused by tobacco, and the economic burden of increased tobacco-related morbidity and mortality runs in the hundreds of billions of dollars.[2] Many smokers, however, remain unaware of the harmful consequences of their tobacco use for themselves, their families, as well as for the larger public. Many others, although knowing about these consequences to themselves and others, still have profound difficulty in quitting because of the addictive nature of nicotine.[3]

There are several ways children and adolescents can be exposed to tobacco. Prenatally, this occurs through maternal smoking or maternal secondhand smoke (SHS) exposure. During childhood or adolescence, active smoking, SHS and thirdhand smoke are all

*Abbreviations:* ADHD, attention-deficit/hyperacti... DNA, deoxyribonucleic acid; ETS, environmental tobac... 5-hydroxytryptamine (serotonin); IQ, intelligence quotient; ... aromatic hydrocarbon; SHS, secondhand smoke; SIDS, su... syndrome; TSNA, tobacco-specific *N*-nitrosamines.

From the "New York University School of Medicine, N... [b]Department of Internal Medicine, University of Washington ... ment of Population Health and Medicine, New York Univ... Medicine, New York, NY; [d]Department of Environmental ... York University School of Medicine, New York, NY; and ... Pediatrics, New York University School of Medicine, New...

Curr Probl Pediatr Adolesc Health Care 2014;44:219-24... ...ent matter ...Inc. ...1016/j.cppeds.2014.03.007 ...olesc Health Care, September 2014



EDITORIALS www.jpeds.com • THE JOURNAL OF PEDIATRICS

Another Reason to Avoid Second-Hand Smoke

Many studies have described the adverse effects of maternal smoking during pregnancy and second-hand smoke (SHS) exposure in early childhood on child health and development.

Maternal smoking in pregnancy has been linked to lower birth weight and decreased head growth as well as increased risk of sudden infant death syndrome. Postnatal SHS exposure has been connected with poorer health outcomes, including increased risk of upper and lower respiratory tract infections, otitis media, asthma, dental caries, hearing loss, and metabolic syndrome. Previous studies have documented a correlation between prenatal and postnatal exposure to tobacco smoke and increased risk of attention deficit-hyperactivity disorder, behavior and conduct disorders, learning disabilities, cognitive deficits, and academic difficulties. Some investigators have argued that socioeconomic and environmental confounders account for the reported associations between SHS exposure and childhood behavior problems, but more recent studies indicate an independent effect for both prenatal and postnatal exposure to tobacco smoke.[1] Very little information is available about the effect of prenatal or postnatal exposure to SHS on motor development.

In this issue of *The Journal*, two articles examine the relationship between SHS exposure and childhood motor development. The study by Eslampidou et al[2] is a prospective cohort study, whereas the study by Yeramaneni et al[3] is a cross-sectional study. Each describes an association between SHS exposure and motor development, but neither can establish a causal relationship.

See related articles, p 246 and p 253

Eslampidou et al[2] explore the relationship between prenatal SHS exposure as reported by maternal questionnaires. Child developmental status was assessed at 18 months of age using the Bayley Scales of Infant Development-III. They found a 3-point decrease in gross motor score per 10 mg/mL increase in urine cotinine in pregnancy. They also described a trend in declining scores in child cognitive and receptive communication scores with increasing urine cotinine levels, but these decreases did not reach a level of statistical significance after adjusting for potential confounders.

In the second study, Yeramaneni et al[3] examine the relationship between SHS exposure and neuromotor function in 404 children age 7-9 years old from the Appalachian region of Ohio between 2009 and 2013. SHS exposure was determined if a household member smoked at least 1 cigarette per day, or the child had a serum cotinine of at least 0.05 ng/mL. Fifty percent of the children were exposed to SHS based on serum cotinine levels. Based on parent report, 37% of the children were exposed to a median of 20 cigarettes per day per household.

This was a community-based participatory, cross-sectional research study originally designed to assess the impact of manganese exposure on the health of the region's children. Multiple variables were evaluated, including lead and manganese levels, parental IQ, parental education, socioeconomic status, and the home environment. Using the several measures of gross motor and fine motor skills, the authors found that children with SHS exposure had diminished visual-motor coordination, and less well developed fine motor integration skills, balance, and strength. Taken together, these studies indicate that prenatal and childhood SHS exposure



ORIGINAL CONTRIBUTION

Association of Pediatric Dental Caries With Passive Smoking

C. Andrew Aligne, MD, MPH
Mark E. Moss, DDS, PhD
Peggy Auinger, MS
Michael Weitzman, MD

**Context** Dental decay is the most common chronic disease of children and it disproportionately affects those living in poverty, but the reasons for this are not clear. Passive smoking may be a modifiable risk factor for dental caries.

**Objective** To examine the relationship between dental caries and serum cotinine levels.

**Design, Setting, and Participants** Cross-sectional data from the Third National Health and Nutrition Examination Survey (1988-1994) of 3531 children aged 4 to 11 years, who had had both dental examinations and a serum cotinine level measurement.

**Main Outcome Measures** Passive smoking defined as serum cotinine levels of 0.2 to 10 ng/mL and caries defined as decayed (unfilled) or filled tooth surfaces.

**Results** Twenty-five percent of the children had at least 1 unfilled decayed tooth surface and 33% had at least 1 filled surface. Fifty-three percent had cotinine levels consistent with passive smoking. Elevated cotinine level was significantly associated with both decayed (odds ratio [OR], 2.1; 95% confidence interval [CI], 1.5-2.9) and filled (OR, 1.4; 95% CI, 1.1-1.8) tooth surfaces in deciduous but not in permanent teeth. This relationship persisted after adjusting for age, sex, race, family income, geographic region, frequency of dental visits, and blood lead level. For dental caries in deciduous teeth, the adjusted OR was 1.8 (95% CI, 1.2-2.7) for the risk of decayed surfaces and 1.4 (95% CI, 1.1-2.0) for filled surfaces. We estimated the population attributable risk from passive smoking to be 27% for decayed and 14% for filled tooth surfaces.

**Conclusions** There is an association between environmental tobacco smoke and risk of caries among children. Reduction of passive smoking is important not only for the

ARTICULO ESPECIAL

Enfermedades pediátricas asociadas al tabaquismo pasivo

J. Ferris Tortajada, J. Alonso López Andreu[1], J. García Castell[2], S. Pérez Tarazona[1], I. Cortell Aznar[1]

*An Esp Pediatr 1998;49:539-547.*

**Introducción**

El tabaquismo constituye uno de los principales problemas sanitarios en los países occidentales. Ocasiona una elevada morbilidad y mortalidad por sus efectos cardiovasculares, cancerígenos, cerebrovasculares y broncopulmonares[1-3]. Genera el 20-25% de la mortalidad global, siendo la principal causa de muerte prematura y evitable[4-6]. El humo producido por la combustión del tabaco contiene más de 4.000 sustancias químicas en fases sólida (macromoléculas o particulas) y gaseosa[7-12]. Aunque el mecanismo de acción de la mayoría de sus componentes asociados a los efectos patógenos no están suficientemente establecidos, sí que se conocen con certeza medio centenar con probado efecto cancerígeno y tóxico sobre los aparatos cardiovascular, respiratorio y reproductor[12-15]. Entre los más peligrosos destacan los siguientes: benceno, cadmio, plomo, polonio 210, 2-naftilamina, 4-nmanobifenil, formaldehído, diversas N-nitrosaminas, benzoantraceno, benzopireno, monóxido de carbono, nicotina, óxido de nitrógeno, tolueno, amoniaco y cianuro de hidrógeno.

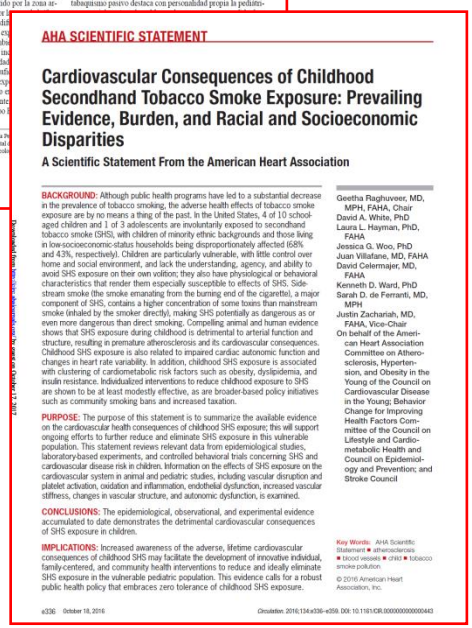
Durante las últimas 2 décadas, diversos estudios han establecido que las personas no fumadoras también presentan una importante morbimortalidad por la exposición al humo tabágico en ambientes cerrados habitados por fumadores (Tabla I)[6-8]. Se les denomina fumadores pasivos, involuntarios o de segunda mano. El humo tabágico constituye el principal contaminante atmosférico de los ambientes domésticos y profesional[13-18]. El humo ambiental es una mezcla formada por: a) humo de la corriente principal exhalada por el fumador; b) humo emitido por la zona ardiente entre las succiones; c) humo emanado por la ... quila" entre las succiones; d) contaminantes dif... del papel del cigarrillo[19-20]. En la tabla II estan ex... tos perjudiciales de la exposición al humo ambi... clasificados en 3 grupos[5,8,13-17]. El grupo A in... ciones del crecimiento y desarrollo, enfermedad... neoplásicas y cardiovasculares en las existe unif... científica que relaciona su desarrollo con la exp... tabáquico. En este grupo se encuentran, tanto e... lesiones (síndrome de la muerte súbita del lactante ... (asma infantil). El grupo

Tabla I Morbimortalidad anual estimada asociada al tabaquismo pasivo en EE.UU. de Norteamérica[6,8]

| | Nº de casos |
|---|---|
| Efecto del desarrollo | |
| Bajo peso al nacimiento | 9.700- 18.600 |
| Síndrome de muerte súbita infantil | 1.900 - 2.700 |
| Efectos respiratorios en niños | |
| Otitis media aguda | 700.000 - 1.600.000 visitas médicos |
| Inducción de asma | 8.000-2.600 nuevos casos |
| Exacerbación de asma | 400.000-1.000.000 |
| Bronquitis o neumonía en < 18 meses | 150.000- 300.000 |
| | 7.500-15.000 hospitalizados |
| | 150 - 212 fallecidos |
| Cáncer | |
| Pulmón | 3.000 fallecidos |
| Cavidad nasal | |
| Cardiovasculares | 35.000 - 62.000 fallecidos |

tos probablemente relacionados aunque sin confirmación precise de más investigaciones. Finalmente, el grupo C incluye las enfermedades posiblemente relacionadas, pero cuyo fundamento científico es menos definitivo.

Entre las poblaciones con mayor riesgo de exposición al tabaquismo pasivo destaca con personalidad propia la pediátri-



AHA SCIENTIFIC STATEMENT

Cardiovascular Consequences of Childhood Secondhand Tobacco Smoke Exposure: Prevailing Evidence, Burden, and Racial and Socioeconomic Disparities

A Scientific Statement From the American Heart Association

Geetha Raghuveer, MD, MPH, FAHA, Chair
David A. White, PhD
Laura L. Hayman, PhD, FAHA
Jessica G. Woo, PhD
Juan Villafane, MD, FAHA
David Celermajer, MD, FAHA
Kenneth D. Ward, PhD
Sarah D. de Ferranti, MD, MPH
Justin Zachariah, MD, FAHA, Vice-Chair
On behalf of the American Heart Association Committee on Atherosclerosis, Hypertension, and Obesity in the Young of the Council on Cardiovascular Disease in the Young; Behavior Change for Improving Health Factors Committee of the Council on Lifestyle and Cardiometabolic Health and Council on Epidemiology and Prevention; and Stroke Council

**BACKGROUND:** Although public health programs have led to a substantial decrease in the prevalence of tobacco smoking, the adverse health effects of tobacco smoke exposure are by no means a thing of the past. In the United States, 4 of 10 school-aged children and 1 of 3 adolescents are involuntarily exposed to secondhand tobacco smoke (SHS), with children of minority ethnic backgrounds and those living in low-socioeconomic-status households being disproportionately affected (68% and 43%, respectively). Children are particularly vulnerable, with little control over home and social environment, and lack the understanding, agency, and ability to avoid SHS exposure on their own volition; they also have physiological or behavioral characteristics that render them especially susceptible to effects of SHS. Side-stream smoke (the smoke emanating from the burning end of the cigarette), a major component of SHS, contains a higher concentration of some toxins than mainstream smoke (inhaled by the smoker directly), making SHS potentially as dangerous as or even more dangerous than direct smoking. Compelling animal and human evidence shows that SHS exposure during childhood is detrimental to arterial function and structure, resulting in premature atherosclerosis and its cardiovascular consequences. Childhood SHS exposure is also related to impaired cardiac autonomic function and changes in heart rate variability. In addition, childhood SHS exposure is associated with clustering of cardiometabolic risk factors such as obesity, dyslipidemia, and insulin resistance. Individualized interventions to reduce childhood exposure to SHS are shown to be at least modestly effective, as are broader-based policy initiatives such as community smoking bans and increased taxation.

**PURPOSE:** The purpose of this statement is to summarize the available evidence on the cardiovascular health consequences of childhood SHS exposure; this will support ongoing efforts to further reduce and eliminate SHS exposure in this vulnerable population. This statement reviews relevant data from epidemiological studies, laboratory-based experiments, and controlled behavioral trials concerning SHS and cardiovascular disease risk in children. Information on the effects of SHS exposure on the cardiovascular system in animal and pediatric studies, including vascular disruption and platelet activation, oxidation and inflammation, endothelial dysfunction, increased vascular stiffness, changes in vascular structure, and autonomic dysfunction, is examined.

**CONCLUSIONS:** The epidemiological, observational, and experimental evidence accumulated to date demonstrates the detrimental cardiovascular consequences of SHS exposure in children.

**IMPLICATIONS:** Increased awareness of the adverse, lifetime cardiovascular consequences of childhood SHS may facilitate the development of innovative individual, family-centered, and community health interventions to reduce and ideally eliminate SHS exposure in the vulnerable pediatric population. This evidence calls for a robust public health policy that embraces zero tolerance of childhood SHS exposure.

Key Words: AHA Scientific Statement ■ atherosclerosis ■ blood vessels ■ child ■ tobacco smoke pollution





Circulation. 2016;134:e336-e359. DOI: 10.1161/CIR.0000000000000443

# Caries dental

ORIGINAL CONTRIBUTION

## Association of Pediatric Dental Caries With Passive Smoking

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**Conclusions** There is an association between environmental tobacco smoke and risk of caries among children. Reduction of passive smoking is important not only for the

TOOTH DECAY IS THE MOST COMmon chronic childhood disease; annual treatment costs in the United States are estimated to be at least $4.5 billion.[1,2] If left untreated, tooth decay can result in substantial morbidity due to pain, dysfunction, poor appearance, and possibly problems with speech development. In the past 50 years, probably as a result of dentifrices and increased levels of fluoride in drinking water, pediatric dental caries occurrence in the United States has declined dramatically; nevertheless, there remain important disparities in caries prevalence within the population.[3,4] In particular, young chil-

**Table 1.** Distribution of Children by Study Variables and by Outcomes of Decayed and Filled Surfaces (Unadjusted Analysis)

| | Deciduous Teeth | | | | | Permanent Teeth | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Unweighted No. of Subjects* | Prevalence of Decayed Surfaces, % | *P* Value† | Prevalence of Filled Surfaces, % | *P* Value† | Unweighted No. of Subjects‡ | Prevalence of Decayed Surfaces, % | *P* Value† | Prevalence of Filled Surfaces, % | *P* Value† |
| Total population | 3531 | 25.4 | | 33.1 | | 2930 | 9.0 | | 18.9 | |
| Serum cotinine level, ng/mL | | | | | | | | | | |
| <0.2§ | 1575 | 18.2 | | 29.2 | | 1340 | 7.4 | | 19.7 | |
| 0.2-10 | 1956 | 31.7 | <.001 | 36.5 | .01 | 1590 | 10.4 | .07 | 18.3 | .59 |
| Poverty status, FPL % | | | | | | | | | | |
| <200 | 2257 | 35.4 | <.001 | 36.7 | .05 | 1863 | 12.4 | <.001 | 19.3 | .81 |
| ≥200§ | 1020 | 15.3 | | 29.4 | | 847 | 5.7 | | 18.6 | |
| Education level of head of household | | | | | | | | | | |
| <High school | 1454 | 40.1 | <.001 | 39.2 | .13 | 1227 | 13.5 | .21 | 20.1 | .81 |
| High school§ | 1103 | 26.9 | | 33.7 | | 910 | 10.8 | | 21.1 | |
| >High school | 930 | 15.5 | <.001 | 29.3 | .17 | 763 | 5.0 | .005 | 16.3 | .14 |
| Race/ethnicity | | | | | | | | | | |
| Non-Hispanic, white§ | 967 | 20.4 | | 33.5 | | 764 | 6.5 | | 19.0 | |
| Non-Hispanic, black | 1097 | 31.7 | <.001 | 25.5 | .003 | 991 | 12.9 | <.001 | 12.5 | .003 |
| Mexican American | 1303 | 41.4 | <.001 | 33.9 | .89 | 1043 | 13.7 | <.001 | 19.3 | .92 |
| Other | 164 | 34.9 | .01 | 40.5 | .28 | 132 | 15.5 | .01 | 28.1 | .12 |
| Region | | | | | | | | | | |
| Northeast§ | 352 | 21.7 | | 33.1 | | 300 | 9.6 | | 23.6 | |
| Midwest | 619 | 20.5 | .73 | 33.6 | .94 | 538 | 6.7 | .29 | 22.7 | .87 |
| South | 1515 | 29.3 | .02 | 30.6 | .52 | 1244 | 10.7 | .69 | 15.9 | .07 |
| West | 1045 | 27.1 | .12 | 35.8 | .53 | 848 | 8.5 | .78 | 16.0 | .13 |
| Sex | | | | | | | | | | |
| Male | 1825 | 24.9 | .61 | 30.6 | .15 | 1449 | 7.8 | .14 | 17.9 | .41 |
| Female§ | 1706 | 25.8 | | 35.7 | | 1481 | 10.2 | | 20.0 | |
| Age, y | | | | | | | | | | |
| 4-7 | 2033 | 25.4 | .95 | 25.2 | <.001 | 1090 | 6.0 | .01 | 6.3 | <.001 |
| 8-11§ | 1498 | 25.3 | | 40.4 | | 1840 | 10.5 | | 25.4 | |
| Sucrose intake, g/d | | | | | | | | | | |
| ≥45.3 | 1523 | 25.0 | .97 | 34.9 | .36 | 1340 | 8.6 | .99 | 20.2 | .32 |
| <45.3§ | 1781 | 25.1 | | 31.3 | | 1449 | 8.6 | | 18.2 | |
| Time since last visit to dentist, d | | | | | | | | | | |
| ≤365§ | 2214 | 20.6 | | 39.7 | | 1948 | 7.6 | | 22.5 | |
| >365 | 1294 | 39.1 | <.001 | 14.1 | <.001 | 964 | 13.8 | .01 | 7.3 | <.001 |
| Blood lead level, µg/dL | | | | | | | | | | |
| ≥10 | 185 | 37.1 | .12 | 22.2 | .003 | 132 | 7.3 | .47 | 12.3 | .09 |
| <10§ | 3337 | 25.0 | | 33.4 | | 2789 | 9.0 | | 19.1 | |

Abbreviation: FPL, Federal poverty level.
*Had at least 1 primary (deciduous) tooth. Totals of less than 3531 are due to missing observations.
†Values correspond to the $\chi^2$ test for the difference in prevalence of caries by study variable category compared with the referent group for each variable.
‡Had at least 1 permanent tooth. Totals of less than 2930 are due to missing observations.
§Indicates referent group.

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# Children with Special Health Care Need's Association of Passive Tobacco Smoke Exposure and Dental Caries: 2007 National Survey of Children's Health

**R Constance Wiener***
Dental Practice and Rural Health, School of Dentistry, Department of Epidemiology, School of Public Health, West Virginia University, USA

**Abstract**

## Table 3

Logistic Regression Odds ratios and Adjusted Odds Ratios for Caries and Passive Smoke Exposure: 2007 National Survey of Children's Health

| | All children Odds (95% CI) Ratios | p-value | CSHCN Odds (95%CI) Ratios | p-value |
|---|---|---|---|---|
| **Unadjusted Passive smoke exposure** | 1.37 (1.23, 1.52) | <.0001 | 1.35 (1.13, 1.62) | 0.0011 |
| **Adjusted Passive Smoke exposure** | 1.27 (1.14, 1.41) | <.0001 | 1.23 (1.02, 1.50) | 0.0352 |
| **Race/ethnicity** | | | | |
| **Non-Hispanic White** | 1.00 (reference) | | 1.00 (reference) | |
| **Non-Hispanic Black** | 1.08 (0.96, 1.22) | 0.0085 | 1.00 (0.78, 1.27) | 0.0214 |
| **Hispanic** | 1.75 (1.05, 1.32) | <.0001 | 1.90 (1.38, 2.60) | 0.0004 |
| **Other** | 1.20 (1.02, 1.41) | 0.6967 | 1.27 (0.94, 1.71) | 0.8734 |
| **Age** | | | | |
| **0-5** | 1.00 (reference) | | 1.00 (reference) | |
| **6-11** | 2.66 (2.32, 3.05) | <.0001 | 2.40 (1.77, 3.24) | <.0001 |
| **12-17** | 1.86 (1.62, 2.14) | 0.0095 | 1.97 (1.46, 2.65) | 0.0198 |
| **Household income** | | | | |
| **Less than 100%FPL** | 1.91 (1.64, 2.22) | <.0001 | 2.27 (1.69, 3.05) | <.0001 |
| **100-199%FPL** | 1.65 (1.44, 1.90) | 0.0002 | 1.75 (1.33, 2.31) | 0.0294 |
| **200-399%FPL** | 1.22 (1.09, 1.38) | 0.0002 | 1/18 (0.90, 1.54) | 0.0058 |
| **400% and above** | 1.00 (reference) | | | |
| **CSHCN** | 1.22 (1.09, 1.36) | 0.0003 | | |

Data Source: Child and Adolescent Health Measurement Initiative (CAHMI). *DRC Indicator Dataset: 2007 National Survey of Children's Health*. Data Resource Center for Child and Adolescent Health, www.childhealthdata.org.

# Desarrollo neuromotor

THE JOURNAL OF PEDIATRICS • www.jpeds.com ORIGINAL ARTICLES

## Secondhand Tobacco Smoke Exposure and Neuromotor Function in Rural Children

Samrat Yeramaneni, MBBS, PhD[1,2], Kim N. Dietrich, PhD[1], Kimberly Yolton, PhD[3], Patrick J. Parsons, PhD[4,5], Kenneth M. Aldous, PhD[4,5], and Erin N. Haynes, DrPH[1]

**Table III. Multivariable associations between logarithm serum cotinine levels and children's neuromotor performance**

| Neuromotor outcomes | Explanatory variable | β (95% CI) | *P* value | Adjusted $R^2$ |
|---|---|---|---|---|
| Dominant hand HRFOT | Serum cotinine | −0.19 (−0.54 to 0.14) | .25 | 0.18 |
| | Age | 2.52 (1.81 to 3.24) | <.0001 | |
| | Sex* | 1.61 (0.30 to 2.92) | .02 | |
| | BPb | −1.50 (−3.03 to 0.03) | .05 | |
| | HMn | −0.49 (−1.27 to 0.28) | .21 | |
| Nondominant hand HRFOT | Serum cotinine | −0.26 (−0.57 to 0.04) | .09 | 0.31 |
| | Age | 2.57 (2.00 to 3.13) | <.0001 | |
| | Sex | 2.86 (1.83 to 3.89) | <.0001 | |
| | Barratt's Education | 0.12 (−0.11 to 0.36) | .30 | |
| | BPb | −1.97 (−3.16 to −0.77) | .003 | |
| | BMn (linear) | 35.29 (4.14 to 66.44) | .02 | |
| | BMn (quadratic) | −7.87 (−15.09 to −1.19) | .02 | |
| Dominant hand PGPT | Serum cotinine | 0.41 (−0.15 to 0.97) | .15 | 0.21 |
| | Age | −4.17 (−5.22 to −3.13) | <.0001 | |
| | BMI | 0.32 (0.05 to 0.60) | .02 | |
| | Barratt's Education | −0.33 (−0.77 to 0.11) | .14 | |
| | BMn (linear) | 0.78 (−3.10 to 4.66) | .69 | |
| | HMn (linear) | 0.24 (−0.84 to 1.32) | .65 | |
| Nondominant hand PGPT | Serum cotinine | 0.71 (0.16 to 1.26) | .01 | 0.19 |
| | Age | −3.95 (−5.01 to −2.90) | <.0001 | |
| | Barratt's Education | −0.34 (−0.79 to 0.10) | .13 | |
| | BMn (linear) | 2.90 (−1.11 to 6.91) | .16 | |
| BOT-2, Total Motor Composite Score | Serum cotinine | −0.64 (−1.13 to −0.16) | .009 | 0.15 |
| | BMI | −0.44 (−0.69 to −0.20) | .003 | |
| | Barratt's Education | 0.33 (−0.05 to 0.71) | .09 | |
| | BPb | 1.18 (−0.71 to 3.07) | .22 | |
| | BMn (linear) | 69.61 (20.80 to 118.43) | .005 | |
| | BMn (quadratic) | −15.63 (−26.41 to −4.85) | .004 | |

*Reference group is female.

# Another Reason to Avoid Second-Hand Smoke

Many studies have described the adverse effects of maternal smoking during pregnancy and second-hand smoke (SHS) exposure in early childhood on child health and development.

natal SHS exposure as reported by maternal questionnaires. Child developmental status was assessed at 18 months of age using the Bayley Scales of Infant Development–III. They found a 3-point decrease in gross motor score per

In this issue of *The Journal*, two articles examine the relationship between SHS exposure and childhood motor development. The study by Evlampidou et al[2] is a prospective cohort study, whereas the study by Yeramaneni et al[3] is a cross-sectional study. Each describes an association between SHS exposure and motor development, but neither can establish a causal relationship.

# Obesidad y riesgo cardiovascular

AHA SCIENTIFIC STATEMENT

**Cardiovascular Consequences of Childhood Secondhand Tobacco Smoke Exposure: Prevailing Evidence, Burden, and Racial and Socioeconomic Disparities**

**A Scientific Statement From the American Heart Association**



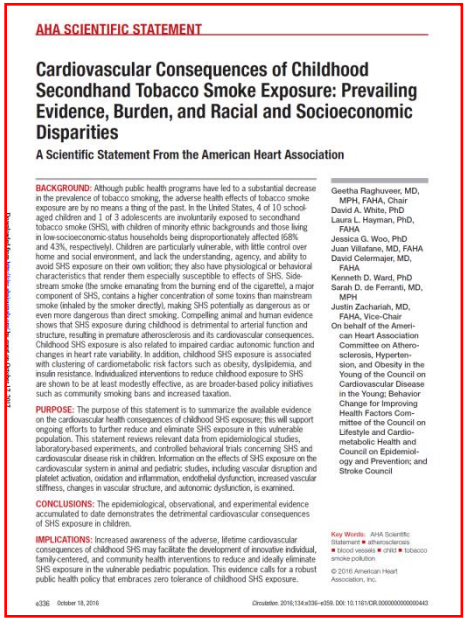
*Circulation.* 2016;134:e336–e359. DOI: 10.1161/CIR.0000000000000443

The Southern California Children's Health Study collected data on current SHS exposure and maternal smoking during pregnancy on 3318 children who were ≈10 years of age at study entry. Both in utero SHS exposure and current SHS exposure were associated with greater subsequent body mass index over an 8-year period spanning from adolescence through young adulthood.[67] Maternal smoking during pregnancy has been reported to result in a 60% greater chance of the child being overweight at 4 years of age.[68] A Swedish cohort study of 5- to 15-year-old children has suggested that parental smoking is associated with a 3% to 4% body mass index increase in children compared with control subjects.[69] In a large study of German children, SHS exposure after birth was significantly associated with overweight status at 6 years of age.[64] The mechanisms behind this association are not well understood. Taken together, results of these studies suggest adverse effects of SHS exposure, including in utero exposure on body mass index, in children.

## Summary

- In utero/postnatal SHS exposure is associated with obesity and cardiovascular risk factors.

# Disfunción cardiovascular



AHA SCIENTIFIC STATEMENT

**Cardiovascular Consequences of Childhood Secondhand Tobacco Smoke Exposure: Prevailing Evidence, Burden, and Racial and Socioeconomic Disparities**

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Geetha Raghuveer, MD, MPH, FAHA, Chair
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Juan Villafane, MD, FAHA
David Celermajer, MD, FAHA
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Sarah D. de Ferranti, MD, MPH
Justin Zachariah, MD, FAHA, Vice-Chair
On behalf of the American Heart Association Committee on Atherosclerosis, Hypertension, and Obesity in the Young of the Council on Cardiovascular Disease in the Young; Behavior Change for Improving Health Factors Committee of the Council on Lifestyle and Cardiometabolic Health and Council on Epidemiology and Prevention; and Stroke Council

**BACKGROUND:** Although public health programs have led to a substantial decrease in the prevalence of tobacco smoking, the adverse health effects of tobacco smoke exposure are by no means a thing of the past. In the United States, 4 of 10 school-aged children and 1 of 3 adolescents are involuntarily exposed to secondhand tobacco smoke (SHS), with children of minority ethnic backgrounds and those living in low-socioeconomic-status households being disproportionately affected (68% and 43%, respectively). Children are particularly vulnerable, with little control over home and social environment, and lack the understanding, agency, and ability to avoid SHS exposure on their own volition; they also have physiological or behavioral characteristics that render them especially susceptible to effects of SHS. Sidestream smoke (the smoke emanating from the burning end of the cigarette), a major component of SHS, contains a higher concentration of some toxins than mainstream smoke (inhaled by the smoker directly), making SHS potentially as dangerous as or even more dangerous than direct smoking. Compelling animal and human evidence shows that SHS exposure during childhood is detrimental to arterial function and structure, resulting in premature atherosclerosis and its cardiovascular consequences. Childhood SHS exposure is also related to impaired cardiac autonomic function and changes in heart rate variability. In addition, childhood SHS exposure is associated with clustering of cardiometabolic risk factors such as obesity, dyslipidemia, and insulin resistance. Individualized interventions to reduce childhood exposure to SHS are shown to be at least modestly effective, as are broader-based policy initiatives such as community smoking bans and increased taxation.

**PURPOSE:** The purpose of this statement is to summarize the available evidence on the cardiovascular health consequences of childhood SHS exposure; this will support ongoing efforts to further reduce and eliminate SHS exposure in this vulnerable population. This statement reviews relevant data from epidemiological studies, laboratory-based experiments, and controlled behavioral trials concerning SHS and cardiovascular disease risk in children. Information on the effects of SHS exposure on the cardiovascular system in animal and pediatric studies, including vascular disruption and platelet activation, oxidation and inflammation, endothelial dysfunction, increased vascular stiffness, changes in vascular structure, and autonomic dysfunction, is examined.

**CONCLUSIONS:** The epidemiological, observational, and experimental evidence accumulated to date demonstrates the detrimental cardiovascular consequences of SHS exposure in children.

**IMPLICATIONS:** Increased awareness of the adverse, lifetime cardiovascular consequences of childhood SHS may facilitate the development of innovative individual, family-centered, and community health interventions to reduce and ideally eliminate SHS exposure in the vulnerable pediatric population. This evidence calls for a robust public health policy that embraces zero tolerance of childhood SHS exposure.

Key Words: AHA Scientific Statement ■ atherosclerosis ■ blood vessels ■ child ■ tobacco smoke pollution





*Circulation*. 2016;134:e336–e359. DOI: 10.1161/CIR.0000000000000443

## CARDIOVASCULAR DYSFUNCTION ASSOCIATED WITH SHS EXPOSURE IN CHILDREN

Cardiovascular effects of key components of cigarettes include

- Nicotine: hemodynamic alterations
- Acrolein: oxidation, inflammation, atherogenesis, hypertension, and arrhythmia
- Crotonaldehyde: plaque instability and thrombosis
- Cadmium: inflammation
- Lead: hypertension
- Particulate matter: arrhythmias and inflammation

## Summary

In utero/childhood SHS exposure can result in

- Endothelial dysfunction
- Increased arterial stiffness
- Increased CIMT
- Autonomic dysfunction
- Late onset arrhythmia

# Dermatitis atópica

FROM THE DERMATOLOGY FOUNDATION

**Association of atopic dermatitis with smoking: A systematic review and meta-analysis**

Robert Kantor, BS,[a] Ashley Kim, BS,[a] Jacob P. Thyssen, MD, PhD,[b] and Jonathan I. Silverberg, MD, PhD, MPH[a,b,c]
*Chicago, Illinois, and Hellerup, Denmark*

***Background:*** Tobacco exposure might be a modifiable risk factor for atopic dermatitis (AD).

***Objective:*** We examine the association between AD and exposure to tobacco smoke.

***Methods:*** We performed a systematic review and meta-analysis of observational studies (n = 86) in MEDLINE, EMBASE, Scopus, and Cochrane Library (1823-2015). Quality of evidence was assessed using the Newcastle-Ottawa Scale (NOS). A meta-analysis was performed using random-effects models to estimate pooled odds ratios (OR). Subset analyses were performed for different ages (children, adult), regions, study designs (cross-sectional, longitudinal), study sizes (<5000, ≥5000), study quality (NOS score <6, ≥6), and amount of smoking (mild, extensive).

***Results:*** A diagnosis of AD was associated with higher odds of active smoking (OR 1.87, 95% confidence interval 1.32-2.63) and exposure to passive smoke (OR 1.18, 95% confidence interval 1.01-1.38), but not maternal smoking during pregnancy (OR 1.06, 95% confidence interval 0.80-1.40). The association between active smoking and AD remained significant in children and adults, all continents studied, and study sizes, but all were cross-sectional designs and had NOS score 6 or greater. Passive smoke was associated with AD in children and adults, cross-sectional studies, South/Central American and African studies, study size less than 5000, and NOS score less than 6.

***Limitations:*** AD severity and distribution were not assessed.

***Conclusions:*** Active and passive exposure to smoke are associated with increased AD prevalence. (J Am Acad Dermatol 2016;75:1119-25.)

***Key words:*** active smoking; atopic dermatitis; eczema; meta-analysis; passive smoking; prenatal smoking; prevalence; systematic review.

Atopic dermatitis (AD) is a chronic inflammatory skin disorder with multifactorial causes that include genetic predisposition, immune dysregulation, and external environmental factors.[1,2] The prevalence of AD has increased over the past

*Abbreviations used:*
AD: atopic dermatitis
CI: confidence interval
NOS: Newcastle-Ottawa Scale
OR: odds ratio

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Mr Kantor and Ms Kim contributed equally to this article.

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Conflicts of interest: None declared.

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## CAPSULE SUMMARY

- Previous studies found conflicting results about whether atopic dermatitis (AD) is associated with tobacco exposure.
- This meta-analysis found that active smoking and passive exposure to smoke were associated with increased prevalence of AD. Maternal smoking during pregnancy was not associated with AD in the children.
- Increased screening and counseling for cessation of smoking appear warranted in patients with AD.

**Table I.** Random-effect models and sensitivity analyses for the association between smoking and atopic dermatitis

| | OR (95% CI) | | |
|---|---|---|---|
| **Study subsets** | **Active smoking, n = 20** | **Passive smoke exposure, n = 66** | **Maternal smoking during pregnancy, n = 23** |
| Overall | **1.87 (1.32-2.63)** | **1.18 (1.01-1.38)** | 1.06 (0.80-1.40) |
| Age | | | |
| Children <18 y | **2.19 (1.34-3.57)** | **1.15 (1.01-1.30)** | 1.02 (0.81-1.27) |
| Adults ≥18 y | **[illegible]** | **[illegible]** | N/A |
| Study design | | | |
| Case-control/cross-sectional | **1.87 (1.32-2.63)** | **1.19 (1.01-1.41)** | 1.14 (0.79-1.65) |
| Cohort/longitudinal | N/A | 1.13 (0.79-1.61) | 0.93 (0.65-1.34) |
| Region | | | |
| North America | **1.66 (1.42-1.93)** | 1.28 (0.94-1.76) | 0.49 (0.15-1.65) |
| South/Central America | N/A | **1.95 (1.55-2.46)** | N/A |
| Asia | **1.74 (1.05-2.88)** | 1.22 (0.98-1.51) | **1.59 (1.28-2.03)** |
| Africa | **8.41 (3.66-19.32)** | **2.23 (1.19-4.19)** | N/A |
| Europe | **1.91 (1.16-3.13)** | 0.96 (0.75-1.22) | 0.94 (0.68-1.10) |
| Study size | | | |
| <5000 | **2.08 (1.31-3.32)** | **1.30 (1.07-1.59)** | 1.37 (0.94-1.95) |
| ≥5000 | **1.68 (1.09-2.53)** | 1.02 (0.81-1.29) | **0.77 (0.58-0.93)** |
| NOS score | | | |
| <6 | N/A | **1.18 (1.11-1.26)** | **1.96 (1.50-2.64)** |
| ≥6 | **1.87 (1.32-2.63)** | 1.11 (0.91-1.36) | 0.88 (0.67-1.16) |
| **Amount of smoking** | **(n = 2)** | **(n = 11)** | **(n = 5)** |
| Mild | **2.68 (1.78-4.04)** | 1.21 (0.81-1.80) | 1.09 (0.68-1.72) |
| Extensive | **2.70 (1.11-6.60)** | 1.08 (0.74-1.59) | 0.74 (0.36-1.52) |

# Physical, Behavioral, and Cognitive Effects of Prenatal Tobacco and Postnatal Secondhand Smoke Exposure

Sherry Zhou,[a] David G. Rosenthal, MD,[b] Scott Sherman, MD, MPH,[c] Judith Zelikoff, PhD,[a,c] Terry Gordon, PhD,[a,d] and Michael Weitzman, MD[a,d,e]

The purpose of this review is to examine the rapidly expanding literature regarding the effects of prenatal tobacco and postnatal secondhand smoke (SHS) exposure on child health and development. Mechanisms of SHS exposure are reviewed, including critical periods during which exposure to tobacco products appears to be particularly harmful to the developing fetus and child. The biological, biochemical, and neurologic effects of the small fraction of identified components of SHS are described. Research describing these adverse effects of both in utero and childhood exposure is reviewed, including findings from both animal models and humans. The following adverse physical outcomes are discussed: sudden infant death syndrome, low birth weight, decreased head circumference, respiratory infections, otitis media, asthma, childhood cancer, hearing loss, dental caries, and the metabolic syndrome. In addition, the association between the following adverse cognitive and behavioral outcomes and such exposures is described: conduct disorder, attention-deficit/hyperactivity disorder, poor academic achievement, and cognitive impairment. The evidence supporting the adverse effects of SHS exposure is extensive yet rapidly expanding due to improving technology and increased awareness of this profound public health problem. The growing use of alternative tobacco products, such as hookahs (a.k.a. waterpipes), and the scant literature on possible effects from prenatal and secondhand smoke exposure from these products are also discussed. A review of the current knowledge of this important subject has implications for future research as well as public policy and clinical practice.

*Curr Probl Pediatr Adolesc Health Care 2014;44:219-241*

## Introduction

More than 1 billion people worldwide smoke tobacco products, and it is predicted that this remarkable number will reach 1.9 billion by 2025.[1] According to the World Health Organization (WHO), there are approximately 6 million deaths per year caused by tobacco, and the economic burden of increased tobacco-related morbidity and mortality runs in the hundreds of billions of dollars.[2] Many smokers, however, remain unaware of the harmful consequences of their tobacco use for themselves, their families, as well as for the larger public. Many others, although knowing about these consequences to themselves and others, still have profound difficulty in quitting because of the addictive nature of nicotine.[2]

There are several ways children and adolescents can be exposed to tobacco. Prenatally, this occurs through maternal smoking or maternal secondhand smoke (SHS) exposure. During childhood or adolescence, active smoking, SHS and thirdhand smoke are all possible means of exposure.

The first evidence statistically linking tobacco smoking with lung cancer appeared in the German journal *Der Tabakgegner* (The Tobacco Opponent) in 1912.[3] Doll and Hill[4] confirmed this finding in 1950 using elegantly developed epidemiologic techniques. These and other findings linking tobacco smoking and adverse health consequences became widely recognized worldwide by the 1964 publication in the U.S. of the seminal report entitled *Smoking and Health: Report of the Advisory Committee to the Surgeon General of the Public Health Service*.[5] It is now authoritatively recognized that there is

*Abbreviations:* ADHD, attention-deficit/hyperactivity disorder; DNA, deoxyribonucleic acid; ETS, environmental tobacco smoke; 5-HT, 5-hydroxytryptamine (serotonin); IQ, intelligence quotient; PAH, polycyclic aromatic hydrocarbon; SHS, secondhand smoke; SIDS, sudden infant death syndrome; TSNA, tobacco-specific *N*-nitrosamines.

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# ¿Hay más?

## SHS Exposure and Hearing Loss

Recently, studies have begun to investigate the association between exposure to SHS and hearing loss.[157] Cigarette smoke damages the entire cochlea, causing hearing loss across the entire frequency spectrum.[158] Smokers are up to twice as likely to experience hearing loss as compared to nonsmokers after adjusting for multiple potential confounders.[159] Prenatal tobacco smoke exposure has been found to be independently associated with higher pure-tone hearing thresholds and a nearly three-fold increase in unilateral low-frequency hearing loss among adolescents.[157,160]

Prenatal smoke exposure is associated with decreased performance on auditory tasks as early as the neonatal period[161–163] and a dose-dependent relationship between SHS exposure and decreased auditory-related tasks.[164,165] In utero exposure, even if the mother quits smoking during the first trimester of pregnancy, may be injurious to the developing auditory system, which also develops during the first trimester.[166] These hearing deficits may contribute to the cognitive and behavioral deficits that persist throughout life.[165]

Although no mechanism has been established to explain the association between hearing loss and SHS exposure,[157] proposed mechanisms include fetal malnourishment due to altered placental architecture,[167] fetal hypoxia due to vasoconstriction,[168] or direct damage by nicotine or other chemicals present in cigarette smoke.[160] Very similar findings have recently been published concerning prenatal tobacco exposure and sensorineural hearing loss.[169]

AHA SCIENTIFIC STATEMENT

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**CONCLUSIONS:** The epidemiological, observational, and experimental evidence accumulated to date demonstrates the detrimental cardiovascular consequences of SHS exposure in children.

**IMPLICATIONS:** Increased awareness of the adverse, lifetime cardiovascular consequences of childhood SHS may facilitate the development of innovative individual, family-centered, and community health interventions to reduce and ideally eliminate SHS exposure in the vulnerable pediatric population. This evidence calls for a robust public health policy that embraces zero tolerance of childhood SHS exposure.

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Key Words: AHA Scientific Statement ■ atherosclerosis ■ blood vessels ■ child ■ tobacco smoke pollution





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# Impacto económico

## ECONOMIC IMPACT OF SHS EXPOSURE

### Summary

Childhood SHS exposure is associated with

- Higher emergency room visits, inpatient use, and medical expenses
- Negative impact on the education system
- Higher rates of behavioral and cognitive adversities
- Increased school absenteeism

# ¿Me olvido de algo?

# Muerte súbita infantil

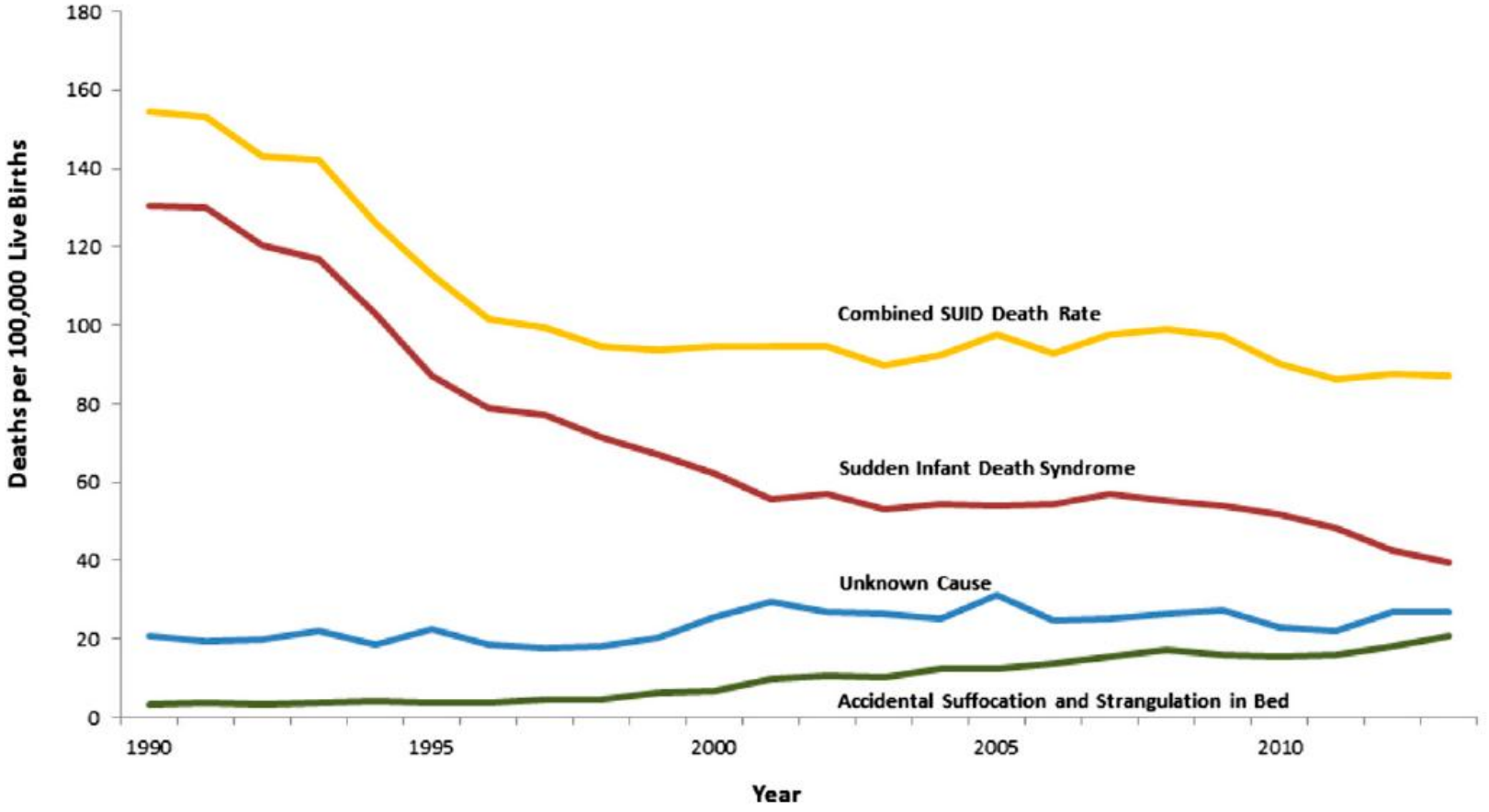
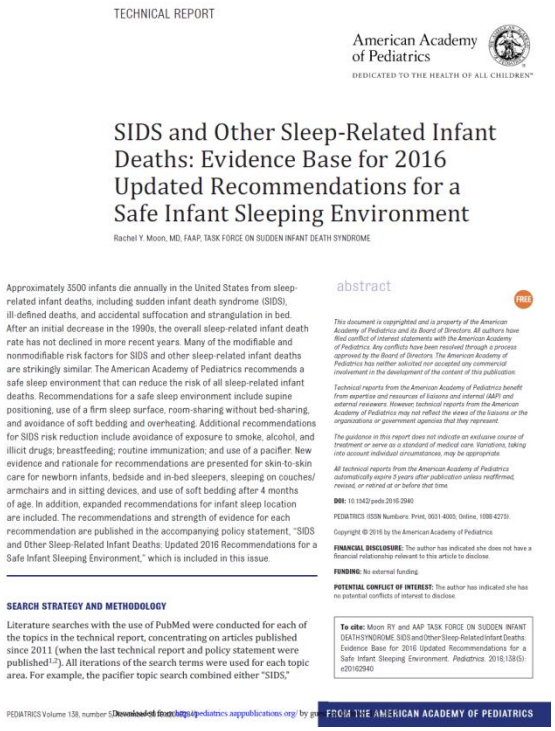



**FIGURE 1**
Trends in SUID by cause, 1990–2013. Source: Centers for Disease Control and Prevention/National Center for Health Statistics, National Vital Statistics System, compressed mortality file. (Figure duplicated from http://www.cdc.gov/sids/data.htm.)



TECHNICAL REPORT

American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN™

# SIDS and Other Sleep-Related Infant Deaths: Evidence Base for 2016 Updated Recommendations for a Safe Infant Sleeping Environment

Rachel Y. Moon, MD, FAAP, TASK FORCE ON SUDDEN INFANT DEATH SYNDROME

Approximately 3500 infants die annually in the United States from sleep-related infant deaths, including sudden infant death syndrome (SIDS), ill-defined deaths, and accidental suffocation and strangulation in bed. After an initial decrease in the 1990s, the overall sleep-related infant death rate has not declined in more recent years. Many of the modifiable and nonmodifiable risk factors for SIDS and other sleep-related infant deaths are strikingly similar. The American Academy of Pediatrics recommends a safe sleep environment that can reduce the risk of all sleep-related infant deaths. Recommendations for a safe sleep environment include supine positioning, use of a firm sleep surface, room-sharing without bed-sharing, and avoidance of soft bedding and overheating. Additional recommendations for SIDS risk reduction include avoidance of exposure to smoke, alcohol, and illicit drugs; breastfeeding; routine immunization; and use of a pacifier. New evidence and rationale for recommendations are presented for skin-to-skin care for newborn infants, bedside and in-bed sleepers, sleeping on couches/armchairs and in sitting devices, and use of soft bedding after 4 months of age. In addition, expanded recommendations for infant sleep location are included. The recommendations and strength of evidence for each recommendation are published in the accompanying policy statement, "SIDS and Other Sleep-Related Infant Deaths: Updated 2016 Recommendations for a Safe Infant Sleeping Environment," which is included in this issue.

abstract

FREE



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## SEARCH STRATEGY AND METHODOLOGY

Literature searches with the use of PubMed were conducted for each of the topics in the technical report, concentrating on articles published since 2011 (when the last technical report and policy statement were published[1–2]). All iterations of the search terms were used for each topic area. For example, the pacifier topic search combined either "SIDS,"

 



To reduce the risk of SIDS, infants should be placed for sleep in the supine position (wholly on the back) for every sleep period by every caregiver until 1 year of age. Side sleeping is not safe and is not advised.

As stated in the AAP clinical report, "skin-to-skin care is recommended for all mothers and newborns, regardless of feeding or delivery method, immediately following birth (as soon as the mother is medically stable, awake, and able to respond to her newborn), and to continue for at least an hour."[130] Thereafter, or when the mother needs to sleep or take care of other needs, infants should be placed supine in a bassinet.

The supine sleep position does not increase the risk of choking and aspiration in infants, even in those with gastroesophageal reflux.

Preterm infants should be placed supine as soon as possible.

Once an infant can roll from supine to prone and from prone to supine, the infant may remain in the sleep position that he or she assumes.

Infants should be placed on a firm sleep surface (eg, a mattress in a safety-approved crib) covered by a fitted sheet with no other bedding or soft objects to reduce the risk of SIDS and suffocation.

A crib, bassinet, portable crib, or play yard that conforms to the safety standards of the Consumer Product Safety Commission (CPSC) is recommended.

Sitting devices, such as car seats, strollers, swings, infant carriers, and infant slings, are not recommended for routine sleep in the hospital or at home, particularly for young infants.

Breastfeeding is associated with a reduced risk of SIDS. The protective effect of breastfeeding increases with exclusivity. Furthermore, any breastfeeding is more protective against SIDS than no breastfeeding.

Smoking during pregnancy, in the pregnant woman's environment, and in the infant's environment should be avoided.

Maternal smoking during pregnancy has been identified as a major risk factor in almost every epidemiologic study of SIDS.[285–288] Smoke in the infant's environment after birth has been identified as a separate major risk factor in a few studies,[286,289] although separating this variable from maternal smoking before birth is problematic. Third-hand smoke refers to residual contamination from tobacco smoke after the cigarette has been extinguished[290]; there is no research to date on the significance of third-hand smoke with regard to SIDS risk. Smoke exposure adversely affects infant arousal[291–297]; in addition, smoke exposure increases the risk of preterm birth and low birth weight, both risk factors for SIDS. The effect of tobacco smoke exposure on SIDS risk is dose-dependent. The risk of SIDS is particularly high when the infant bed-shares with an adult smoker (OR: 2.3–21.6), even when the adult does not smoke in bed.[89,90,191,200,201,206,212,298] It is estimated that one-third of SIDS deaths could be prevented if all maternal smoking during pregnancy was eliminated.[299,300] The AAP supports the elimination of all tobacco smoke exposure, both prenatally and environmentally.

Se calcula que se podrían prevenir la tercera parte de los SMSI si se eliminara todo el tabaquismo materno.

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# Objetivos docentes

- Al final de la presentación, el asistente será capaz sostener con pruebas que el tabaquismo pasivo en niños:
  - Aumenta ***la caries dental***
  - Se asocia a menor ***desarrollo neuromotor***
  - Aumenta el riesgo de ***obesidad y riesgos cardiovasculares***
  - Se asocia a ***disfunción cardiovascular***
  - Parece asociarse a ***dermatitis atópica***
  - ***Es caro***
  - En general, ***es malo para todo***.

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