904 Environmental Tobacco Smoke Exposure

Definition/Cut-off Value

Environmental tobacco smoke (ETS) exposure is defined (for WIC eligibility purposes) as exposure to smoke from tobacco products inside the home (1, 2, 3).* ETS is also known as passive, secondhand, or involuntary smoke.

** See Clarification for background information*

Participant Category and Priority Level

Category	Priority
Pregnant Women	I
Breastfeeding Women	I
Non-Breastfeeding Women	III,IV,V, or VI
Infants	I
Children	III

Justification

ETS is a mixture of the smoke given off by a burning cigarette, pipe, or cigar (sidestream smoke), and the smoke exhaled by smokers (mainstream smoke). ETS is a mixture of about 85% sidestream and 15% mainstream smoke (4) made up of over 4,000 chemicals, including Polycyclic Aromatic Hydrocarbons (PAHs) and carbon monoxide (5). Sidestream smoke has a different chemical make-up than main-stream smoke. Sidestream smoke contains higher levels of virtually all carcinogens, compared to mainstream smoke (6). Mainstream smoke has been more extensively researched than sidestream smoke, but they are both produced by the same fundamental processes.

ETS is qualitatively similar to mainstream smoke inhaled by the smoker. The 1986 Surgeon General's report: *The Health Consequences of Involuntary Smoking. A Report of the Surgeon General* concluded that ETS has a toxic and carcinogenic potential similar to that of the mainstream smoke (7). The more recent 2006 Surgeon General's report, *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General,* reaffirms and strengthens the findings of the 1986 report, and expands the list of diseases and adverse health effects caused by ETS (8).

ETS is a known human carcinogen (2). Women who are exposed to ETS are at risk for lung cancer and cardiovascular diseases (9). Prenatal or postnatal ETS exposure is related to numerous adverse health outcomes among infants and children, including sudden infant death syndrome (SIDS) (10, 11), upper respiratory infections (12), periodontal disease (13), increased severity of asthma/wheezing (12), metabolic syndrome (14), decreased cognitive function (15), lower birth weight and smaller head circumference (16). Infants born to women exposed to ETS during pregnancy have a small decrease in birth weight and a slightly increased risk of intrauterine growth retardation compared to infants of unexposed women (17).

Studies suggest that the health effects of ETS exposure at a young age could last into adulthood. These include cancer (18), specifically lung cancer (19, 20), and cardiovascular diseases (14, 21, 22,). There is strong evidence that ETS exposure to the fetus and/or infant results in permanent lung damage (23, 24, 25, 26).

ETS exposure increases inflammation and oxidative stress (27, 28, 29). Inflammation is associated with asthma (30), cardiovascular diseases (31, 32), cancer (33), chronic obstructive pulmonary disease (34), and metabolic syndrome (14, 35). PAHs are the major class of compounds that contribute to the ETS-related adverse health outcomes. These compounds possess potent carcinogenic and immunotoxic properties that aggravate inflammation.

Oxidative stress is a general term used to describe the steady state of oxidative damage caused by highly reactive molecules known as free radicals. The free radicals can be generated both during the normal metabolic process and from ETS and other environmental pollutants. When free radicals are not neutralized by antioxidants, they can cause oxidative damage to the cells. This damage has been implicated in the cause of certain diseases. ETS provokes oxidant damage similar to that of active smoking (36).

Antioxidants may modulate oxidative stress-induced lung damage among both smokers and non-smokers (22, 27-29, 37-40). Fruits and vegetables are the major food sources of antioxidants that may protect the lung from oxidative stress (1). Research indicates that consuming fruits and vegetables is more beneficial than taking antioxidant supplements (1). This suggests that other components of fruits and vegetables may be more relevant in protecting the lung from oxidative stress. Dietary fiber is also thought to contribute to the beneficial health effects of fruits and vegetables (1).

The Institute of Medicine (IOM) reports that an increased turnover in vitamin C has been observed in nonsmokers who are regularly exposed to tobacco smoke (41). The increased turnover results in lowered vitamin C pools in the body.

Although there are insufficient data to estimate a special requirement for non-smokers regularly exposed to ETS, the IOM urges those individuals to ensure that they meet the Recommended Dietary Allowance for vitamin C (36, 41).

The WIC food package supplements the participant intake of vitamin C. In addition, many WIC State Agencies participate in the WIC Farmers' Market Nutrition Program, which provides coupons for participants to purchase fresh fruits and vegetables. WIC Program benefits also include counseling to increase fruit and vegetable consumption, and to promote a healthy lifestyle, such as protecting participants and their children from ETS exposure. WIC staff may also make appropriate referrals to participants, and/or their caregivers, to other health and social services, such as smoking cessation programs.

Clarification

In a comprehensive scientific report, the Surgeon General concluded that there is no risk-free level of exposure to secondhand smoke (8). However, for the purpose of risk identification, the definition used for this risk criterion is based on the Centers for Disease Control and Prevention (CDC) Pediatric Nutrition Surveillance System (PedNSS) and the Pregnancy Nutrition Surveillance System (PNSS) questions to determine Environmental Tobacco Smoke (ETS) exposure:

- Does anyone living in your household smoke inside the home? (infants, children)
- Does anyone else living in your household smoke inside the home? (women)

Because the definition used by other Federal agencies for ETS exposure is specific to "inside the home" and has been validated (3), the definition used for WIC eligibility must also be as specific. In addition, FNS encourages the use of the PedNSS and PNSS ETS exposure questions for WIC nutrition assessment.

There are other potential sources of ETS exposure, such as work and day care environments. However, no other validated questions/definitions could be found that were inclusive of other environments and applicable to WIC.

References

1. Lesley Butler, et al. RISC/WIC Report on Environmental Tobacco Smoke Exposure. February 2006. Unpublished.
2. Respiratory Health Effects of Passive Smoking (Also Known as Exposure to Secondhand Smoke or Environmental Tobacco Smoke ETS). U.S. Environmental Protection Agency, Office of Research and Development, Office of Health and Environmental Assessment, Washington, DC, EPA/600/6-90/006F, 1992. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2835 Accessed March 2006.
3. Pirkle, JL, KM Flegal, DJ Brody, RA Etzel, and KR Maurer. Exposure of the U.S. Population to Environmental Tobacco Smoke. The Third National Health and Nutrition Examination Survey, 1988 to 1991. JAMA; 1996; 275, 16; 1233-1240.
4. Witschi, H, JP Joad, and KE Pinkerton. The toxicology of environmental tobacco smoke. Annu. Rev. Pharmacol. Toxicol. 1997; 37: 29-52.
5. Seifert, JA, CA Ross, and JM Norris. Validation of a five-question survey to assess a child's exposure to environmental tobacco smoke. Ann. Epidemiol. 2002; 12:273-277.
6. Adams, JD, KJ O'Mara-Adams, and D Hoffmann. Toxic and carcinogenic agents in undiluted main-stream smoke and sidestream smoke of different types of cigarettes. Carcinogenesis 1987-8:729-731.
7. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health, 1986. DHHS Publication No. (CDC) 87-8398.
8. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General—Executive Summary. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, Coordinating Center for Health Promotion, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.
9. National Cancer Institute. Health Effects of Exposure to Environment Tobacco Smoke. Smoking and Tobacco Control Monograph No. 10 (PDF - 71k). Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 1999. NIH Pub. No. 99-4645. Accessed: March 2006.
10. Dybing, E., and T Sanner. Passive smoking, sudden infant death syndrome (SIDS) and childhood infections. Hum. Exp. Toxcol. 1999; 18:202-205.

11. Klonoff-Cohen, HS, SL Edelstein, ES Lefkowitz, IP Srinivasan, D Kaegi, JC Chang, and KJ Wiley. The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome. JAMA 1995; 273:795-798.
12. Cook, DG, and DP Strachan. Health effects of passive smoking-10: Summary of effects of parental smoking on the respiratory health of children and implications for research. Thorax 1999; 54:357-366.
13. Aligne, CA, ME Moss, P Auinger, and M Weitzman. Association of pediatric dental caries with passive smoking. JAMA 2003; 289:1258-1264.
14. Weitzman, M, S Cook, P Auinger, TA Florin, S Daniels, M Nguyen, and JP Winickoff. Tobacco smoke exposure is associated with the metabolic syndrome in adolescents. Circulation 2005; 112:862-869.
15. Yolton, K, K Dietrich, P Auinger, BP Lanphear, and R Hornung. Exposure to environmental tobacco smoke and cognitive abilities among U.S. children and adolescents. Environ. Health Perspect. 2005; 113:98-103.
16. Perera, FP, V Rauh, RM Whyatt, WY Tsai, JT Bernert, YH Tu, H Andrews, J Ramirez, L Qu, and D Tang. Molecular evidence of an interaction between prenatal environmental exposures and birth outcomes in a multiethnic population. Environ. Health. Perspect. 2004; 112:626-630.
17. Women and Smoking: A Report of the Surgeon General – 2001. http://www.cdc.gov/tobacco/sgr/sgr_forwomen/index.htm.
18. Tredaniel, J, P Boffetta, J Little, R Saracci, and A Hirsch. Exposure to passive smoking during pregnancy and childhood, and cancer risk: the epidemiological evidence. Paediatr. Perinat. Epidemiol. 1994; 8:233-255.
19. Tredaniel, J, P Boffetta, R Saracci, and A Hirsch. Exposure to environmental tobacco smoke and risk of lung cancer: the epidemiological evidence. Eur. Respir. J. 1994; 7:1877-1888.
20. Janerich, DT, WD Thompson, LR Varela, P Greenwald, S Chorost, C Tucci, MB Zaman, MR Melamed, M Kiely, and MF McKneally. Lung cancer and exposure to tobacco smoke in the household. New Engl. J. Med. 1990; 323:632-636.
21. Moffatt, RJ, BA Stamford, and KD Biggerstaff. Influence of worksite environmental tobacco smoke on serum lipoprotein profiles of female nonsmokers. Metabolism 1995; 44:1536-1539.
22. Moskowitz, WB, M Mosteller, RM Schieken, R Bossano, JK Hewitt, JN Bodurtha, and JP Segrest. Lipoprotein and oxygen transport alterations in passive smoking preadolescent children. The MCV Twin Study. Circulation 1990; 81:586-592.
23. Masi, MA, JA Hanley, P Ernst, and MR Becklake. Environmental exposure to tobacco smoke and lung function in young adults. Am. Rev. Respir. Dis. 1988; 138:296-299.
24. Upton, MN, GC Watt, G Davey Smith, A McConnachie, and CL Hart. Permanent effects of maternal smoking on offsprings' lung function. Lancet 1998; 352:453.
25. Svanes, C, E Omenaas, D Jarvis, S Chinn, A Gulsvik, and P Burney. Parental smoking in childhood and adult obstructive lung disease: results from the European Community Respiratory Health Survey. Thorax 2004; 59:295-302.

26. Grant, Stephen G. Qualitatively and quantitatively similar effects of active and passive maternal tobacco smoke on in utero mutagenesis at the HPRT locus. BMC Pediatrics 2005, 5:20, doi: 10.1186/1471-2431-5-20.

27. Block, G, M Dietrich, EP Norkus, JD Morrow, M Hudes, B Caan, and L Packer. Factors associated with oxidative stress in human populations. Am. J. Epidemiol. 2002; 156:274-285.

28. Morrow, JD, and LJ Roberts, 2nd. Mass spectrometric quantification of F2-isoprostanes in biological fluids and tissues as measure of oxidant stress. Methods Enzymol 1999; 300:3-12.

29. Panagiotakos, DB, C Pitsavos, C Chrysohoou, J Skoumas, C Masoura, P Toutouzas, and C Stefanadis. Effect of exposure to secondhand smoke on markers of inflammation: the ATTICA study. Am. J. Med. 2004; 116:145-150.

30. Leem JH, Kim JH, Lee KH, Hong Y, Lee KH, Kang D, Kwon HJ. Asthma attack associated with oxidative stress by exposure to ETS and PAH.
J. Asthma. 2005 Jul-Aug; 42(6):463-7. PMID: 16293541.

31. Panagiotakos DB, Pitsavos C, Chrysohoou C, Skoumas J, Masoura C, Toutouzas P, Stefanadis C; ATTICA study. Effect of exposure to secondhand smoke on markers of inflammation: the ATTICA study. Am. J. Med. 2004 Feb 1; 116(3):145-50. PMID: 14749157.

32. Ambrose JA, Barua RS. The pathophysiology of cigarette smoking and cardiovascular disease: an update. J. Am. Coll. Cardiol. 2004 May 19; 43(10):1731-7. Review. PMID: 15145091.

33. Sinn DD, Man SF, McWilliams A, Lam S. Progression of airway dysplasia and C-reactive protein in smokers at high risk of lung cancer. Am. J. Respir. Crit. Care Med. 2006 Mar 1:173(5): 535-9. Epub 2005 Dec 9. PMID: 16339918.

34. Bartal M. COPD and tobacco smoke. Monaldi Arch Chest Dis. 2005 Dec; 63(4):213-25. Review. PMID: 16454221.

35. Haffner SM. The metabolic syndrome: inflammation, diabetes mellitus, and cardiovascular disease. Am. J. Cardiol. 2006 Jan 16; 97(2A):3A-11A. Epub 2005 Dec 5. Review. PMID: 16442931.

36. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium and Carotenoids (2000) Institute of Medicine, the National Academy of Science.

37. Smit, HA. Chronic obstructive pulmonary disease, asthma and protective effects of food intake: from hypothesis to evidence? Respir. Res. 2001; 2:261-264.

38. Burney, P. The origins of obstructive airways disease. A role for diet? Am J Respir Crit Care Med 1995; 151:1292-1293.

39. MacNee, W. Oxidants/antioxidants and COPD. Chest 2000; 117:303S-317S.

40. Altose, MD. Approaches to slowing the progression of COPD. Curr. Opin. Pulm. Med. 2003; 9:125-130.

41. Dietary Reference Intakes: The essential Guide to Nutrient Requirements (2006) Otten, JJ, Hellwig, JP, Meyers, LD, ed., Institute of Medicine of the National Academies. The National Academies Press, Washington D.C.