California Environmental Protection Agency (Cal/EPA)

Questions and Answer Document Regarding the Cal/EPA's "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant" Report

1. Why did we produce this report?

Cal/EPA has historically been concerned with the health effects of environmental tobacco smoke (ETS) and produced this report to meet all of the regulatory requirements necessary for the formal identification of ETS as a toxic air contaminant (TAC). ETS was entered into the process for formal identification as a TAC in California in recognition of its significant and many adverse health effects and, in particular, its impacts on infants and children. This report was the first one conducted after the implementation of the Children's Health Protection Act (SB 25) which requires consideration of special susceptibilities of children.

2. Cal/EPA produced a widely cited report on secondhand smoke in 1997. Why didn't we just use that report?

The 1997 report was a comprehensive evaluation of the ETS health effects literature up to that point in time. However, it did not fulfill all of the legal requirements for formal identification as a TAC, especially in terms of exposure. In 1999, Senate Bill 25 (Escutia) was enacted to require ARB to assess exposure patterns and adverse health effects among susceptible infants and children. Furthermore, the law requires that all available evidence be considered; thus, it was necessary to update the 1997 report.

3. What was the process?

The 2005 Cal/EPA ETS report was developed over a four year time period. The Air Resources Board (ARB) began the process by requesting pertinent ETS information from the public, such as ETS exposure and adverse effects on health. Next, the Air Resources Board (ARB) and Office of Environmental Health Hazard Assessment (OEHHA) both initiated their own extensive ETS literature reviews. Once the ETS report was drafted, it was released for public review in December, 2003. A public workshop was held to discuss the report during the formal comment period. After receiving public comments, ARB and OEHHA carefully reviewed all comments, incorporated new information, and revised the report where appropriate.

After the comment period and public workshop, the report was then submitted to the Scientific Review Panel (SRP) on Toxic Air Contaminants to begin the peer review process. The SRP held its first public meeting to discuss the report on November 30, 2004. Because of extensive comments by the SRP, ARB and

OEHHA staff submitted three separate ETS report drafts to be considered at subsequent SRP meetings held on January 6, March 14, and June 24, 2005. The SRP agreed that ARB's and OEHHA's conclusions were scientifically sound, and ultimately approved the June 2005 draft of the report, prepared its "scientific findings" and submitted the findings to ARB. The final SRP approved report was then released to the public on September 29, 2005.

4. What are the health effects of ETS?

After reviewing the epidemiological and toxicological literature on ETS and chemical constituents of ETS, Cal/EPA has determined, based on a weight-of-evidence approach, that there is sufficient evidence that exposure to ETS is associated with a number of adverse health effects. These health effects are provided in Table 1. In addition to its effects on adults, ETS exposure increases the risk for many health effects in infants and children including low birth weight, pre-term delivery, sudden infant death syndrome, respiratory tract and ear infections, and asthma.

5. What is new in the 2005 Cal/EPA report?

For the Part A exposure assessment, ARB presented the first ever estimation of ETS emissions in California, as well as the latest statistics regarding smoking prevalence. ARB also conducted a novel ambient nicotine monitoring program at various outdoor public venues. The data generated are the first California near-source ETS outdoor exposure measurements. ARB staff also estimated ETS concentrations in urban settings to add to the existing body of exposure information.

For Part B, new findings of health effects since our Cal/EPA 1997 report (italicized in Table 1) include causal associations between ETS exposure and induction and exacerbation of asthma in adolescents and adults, pre-term delivery, altered vascular properties (associated with heart attack risk), and breast cancer in younger, primarily premenopausal women.

ETS has been linked previously to induction of asthma in children. Newer studies on adolescents and adults expand that link to these older age groups. While ETS has been linked previously to low birth weight, new evidence also indicates ETS exposure is also linked to pre-term delivery. Premature babies and low birth weight babies are at higher risk for a number of health problems including infant mortality.

The finding of a causal association with breast cancer in younger women is generating much interest. We discuss some of the issues surrounding our finding below.

TABLE 1
HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Effects Causally Associated with ETS Exposure

Developmental Effects
Fetal growth: Low birthweight and decrease in birthweight
Sudden Infant Death Syndrome (SIDS)
Pre-term delivery

Respiratory Effects
Acute lower respiratory tract infections in children (*e.g.,* bronchitis and pneumonia)
Asthma induction and exacerbation in children *and adults*
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children

Carcinogenic Effects
Lung cancer
Nasal sinus cancer
Breast cancer in younger, primarily pre-menopausal women

Cardiovascular Effects
Heart disease mortality
Acute and chronic coronary heart disease morbidity
Altered vascular properties

6.a. Why do the findings on breast cancer in the OEHHA document differ from those of the 2004 report of the U.S. Surgeon General and IARC (2004)?

The 2005 CalEPA report is based on more recent studies than the Surgeon General's report (U.S. DHHS, 2004). The Surgeon General's report relies on essentially the same data as the 1997 CalEPA report, which did not find a causal association for breast cancer. The 2005 CalEPA report considered many more studies published between 2000 and 2005 than the Surgeon General's report. Similarly, the IARC report evaluated few studies published between 2000 and 2002 and no studies published after 2002.

6b. How can OEHHA be confident of their conclusion that ETS exposure increases the risk of breast cancer in younger/primarily premenopausal women?

We have a high level of confidence in our conclusion. OEHHA uses a weight-of-evidence approach to evaluating the epidemiological literature (described in Part B, Chapter 1). Many investigators split out results for premenopausal and postmenopausal women in their studies because of known differences in disease prognosis by menopausal status. Studies that evaluated premenopausal women consistently show elevated breast cancer risks in younger women, with many studies showing statistically significant elevated risks. Several studies showed evidence of dose-response (increasing risk with increasing exposure). Meta analysis (pooling the results together in a single analysis) of the available studies indicates significant elevations in risk, which are higher for breast cancer diagnosed in younger, primarily premenopausal women. Studies that did the most thorough exposure assessments had the highest risks. In contrast to the findings in younger women, studies which reported statistics for women diagnosed with breast cancer after menopause did not appear to show increased risk.

The studies looked at other factors which might explain the elevated breast cancer risk (confounding factors). But these other factors generally had little impact on the level of association between ETS and breast cancer. Most newer studies accounted for the known confounding from reproductive history, alcohol consumption, oral contraceptive use, socioeconomic status, and family history. Furthermore, it is unlikely that bias or confounding would produce an association in younger (mostly premenopausal), but not older (postmenopausal) women within the same studies.

We also note that tobacco smoke contains many chemical carcinogens, including chemicals that cause breast cancer in animals, and that these compounds reach and damage DNA in breast tissue as a result of direct smoking or ETS exposures. All this evidence leads us to the conclusion that ETS exposure contributes to human breast cancer.

For additional technical details on the breast cancer findings, see pages 7-76 to 7-119 in Part B, and response to comments pp.209-230, 239-250. 255-262, and 263-281. in Part C.

7. What do you anticipate will be the next steps?

ARB will conduct a public Board hearing on January 26, 2006, to consider the adoption of a regulatory amendment to identify ETS as a TAC.

8. Who should we contact if we have additional questions?

For additional questions on health effects, please contact Melanie Marty, Ph.D., Chief, Air Toxicology and Epidemiology Branch, at (510) 622-3150. For additional questions on exposure assessment, please contact Ms. Janette Brooks, Chief, Air Quality Measures Branch, at (916) 322-7072.