

February 20, 2007

e-mail attachment to David Gordon, Burbank City Council from Melanie Marty, Ph.D., Chief Air Toxicology and Epidemiology Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency

Dear Mr. Gordon,

Thank you very much for your inquiry regarding our document on environmental tobacco smoke. The epidemiology studies of health effects from exposure to ETS assessed exposure by questions regarding living or working with smokers. In some cases the studies asked about the extent of smoking going on around the nonsmoking subjects, and in still others measurements of nicotine or cotinine in urine were used to differentiate exposure to ETS. It is the concentration of smoke constituents to which one is exposed that drives the health risk rather than the location of exposure (indoors versus outdoors). The measurements of exposures from outdoor smoking conducted by the California Air Resources Board for the listing of ETS as a toxic air contaminant, which were some of the first and most extensive of their type, showed levels of outdoor exposure that were similar to those found in studies that found an association between ETS exposure and exacerbation of asthma. For example, and perhaps most relevant to your question, the paper by Eisner et al., 2001<sup>1</sup> which uses a chemical marker to identify the level of exposure along with a validated history and includes individuals with outside only exposure finds approximately a doubling of reported aggravation of respiratory symptoms, sensory irritation, and extra bronchodilator use in those who had "low level exposure" to ETS. They defined low level exposure as a nicotine concentration of .05mcg/m<sup>3</sup> or less and compared those people with this level of exposure to those with none. The median levels of exposure noted in those with outdoor only exposure was .03mcg/m<sup>3</sup> and was as high as .08 mcg/m<sup>3</sup> in some. In the Eisner study, the median nicotine exposure in the highest exposed was 0.61 mcg/m<sup>3</sup>, a group with reported six fold increase in respiratory symptoms and bronchodilator use. The ARB monitoring results (summarized below in table V-3 from Part A of the document) found outdoor smoking areas with one hour measurements of nicotine as high as 4.6 mcg/m<sup>3</sup> and eight hour measurements as high as 3.1 mcg/m<sup>3</sup>. While one might expect that the time spent in these areas might be limited, the levels are clearly substantially higher than median levels of exposure in the Eisner study that are associated with symptoms and increased medication use in asthmatics. These respiratory symptoms are acute responses and may not require prolonged exposures.

Of particular concern, children may be in outdoor settings where smoking is occurring and cannot limit their exposure by leaving the premises, as can adults. Children are disproportionately affected by asthma with an estimated 14.3 million California children affected in 2003<sup>2</sup>. Asthma is widely considered to be amongst the most serious chronic

---

<sup>1</sup> Mark D. Eisner, Patricia P. Katz, Edward H. Yelin, S. Katharine Hammond, and Paul D. Blanc. Measurement of Environmental Tobacco Smoke Exposure among Adults with Asthma. *Environ Health Perspect* 109:809-814 (2001)

<sup>2</sup> California Department of Health Services, <http://www.dhs.ca.gov/ps/cdic/caphi/>

health problems children in California face today. Nationally, the CDC has documented that despite a major decrease in exposure, children's geometric mean cotinine measurements (a biomarker for tobacco smoke exposure) are still higher than adults<sup>3</sup>. The highest levels were those in ages 3-11 years (younger children were not tested).

A characteristic of a healthy cardiovascular system and the associated autonomic nervous system is a high level of heart rate variability (HRV). Measures of decreased HRV have been associated with increased risk of heart failure (Nolan *et al.*, 1998<sup>4</sup>). Pope *et al.* (2001<sup>5</sup>) examined changes in both time- and frequency-domain measures of HRV in 16 healthy adults during alternating two-hour periods of exposure to ETS or room air in an airport's smoking and nonsmoking areas. Ambulatory electrocardiograph monitors collected data on all participants during the eight hour experiment for analysis of HRV. Over the eight hour period, nicotine and RSP levels were in the ranges 21-53  $\mu\text{g}/\text{m}^3$  and 41-166  $\mu\text{g}/\text{m}^3$ , respectively, in the smoking area, and 0-2  $\mu\text{g}/\text{m}^3$  and 12-43  $\mu\text{g}/\text{m}^3$ , respectively, in the nonsmoking area.

One measure, the standard deviation of normal-to-normal beat intervals (SDNN), correlated most highly with overall measures of HRV and so was used to examine the effect of ETS exposure on HRV. Among six models controlling for various covariates, all ETS exposure variables were negatively and significantly ( $p < 0.05$ ) correlated with SDNN. Thus the overall effect of ETS exposure in this study was a decrease in cardiac autonomic function, as measured by HRV that reversed upon cessation of exposure. The acute effects of ETS on HRV could put susceptible individuals at higher risk of a cardiovascular event (e.g., a myocardial infarction). Although the levels of exposure in this study are higher than the ARB outdoor levels this is evidence of an acute response to a short term exposure that is associated with an increase in heart attack risk. The levels of nicotine (21-53  $\mu\text{g}/\text{m}^3$ ), used as a marker for the level of ETS exposure, associated with these changes in this study are only about five to ten times that measured for one hour exposures in the outdoor smoking area at the amusement park in the ARB study. This would appear to provide a slim margin of safety for those at risk for a cardiovascular event, e.g., those with heart disease. Similarly, Moffatt *et al.*, 2004<sup>6</sup> found a six hour exposure to ETS with an associated nicotine air concentration of 16  $\mu\text{g}/\text{m}^3$  produced a decrease in plasma high-density lipoprotein cholesterol (HDL-C). Similarly, Otsuka *et al.*, demonstrated a decrease in coronary flow velocity reserve (a significant predictor of cardiac events such as myocardial infarction and death) after only a thirty minute

---

<sup>3</sup> Third National Report on Human Exposure to Environmental Chemicals, U.S. Centers for Disease Control, 2005  
<http://www.cdc.gov/exposurereport/3rd/pdf/thirdreport.pdf>

<sup>4</sup> Nolan J, Batin PD, Andrews R, Lindsay SJ, Brooksby P, Mullen M, Baig W, Flapan AD, Cowley A, Prescott RJ, Neilson JM, Fox KA (1998). Prospective study of heart rate variability and mortality in chronic heart failure: results of the United Kingdom heart failure evaluation and assessment of risk trial (UK-heart). *Circulation* 98(15):1510-6.

<sup>5</sup> Pope CA 3<sup>rd</sup>, Eatough DJ, Gold DR, Pang Y, Nielsen KR, Naith P, Verrier RL, Kanner RE (2001). Acute exposure to environmental tobacco smoke and heart rate variability. *Environ Health Perspect* 109(7):711-6.

<sup>6</sup> Moffatt RJ, Chelland SA, Pecott DL, Stamford BA (2004). Acute exposure to environmental tobacco smoke reduces HDL-C and HDL2-C. *Prev Med* 38(5):637-41.

exposure to passive smoke<sup>7</sup>. These last three studies are examples of health effects induced by short-term exposures to ETS that are somewhat higher but still relevant to outdoor exposures.

The findings reported in our document “Health Effects of Environmental Tobacco Smoke” and of the Scientific Review Panel (as well as the U.S. Surgeon General) include conclusive evidence of ETS exposure causing various forms of cancer. It is generally accepted that for carcinogens there is no threshold below which there is zero risk. This underlies the conclusions of the U.S. Surgeon General in his 2006 report (“The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General”) in which it is clearly stated “the scientific evidence indicates that there is no risk-free level of exposure to secondhand smoke”.

Finally, it should be noted that urban ambient air pollution, including traffic-related air pollution, has been associated with a wide-range of health effects including exacerbation of asthma and heart disease. Environmental tobacco smoke has many chemical similarities to traffic-related air pollution, and smoking outdoors may be the source of higher near-source exposures to some important air pollutants (CO, particulate matter, carcinogenic toxic air contaminants, respiratory irritants such as acrolein) than the background levels of air pollutants from traffic.

I hope that this information is helpful in your deliberations. Please feel free to contact this office if there is further information that you need.

---

<sup>7</sup> Otsuka R, Watanabe H, Hirata K, Tokai K, Muro T, Yoshiyama M, Takeuchi K, Yoshikawa J (2001). Acute effects of passive smoking on the coronary circulation in healthy young adults. JAMA 286(4):436-41.

**Table V-3**

**Results of ARB Nicotine Air Monitoring Adjacent to Outdoor Smoking Areas**

Site Tested	8-hour Data	Concentration ( $\mu\text{g}/\text{m}^3$ )	Cigarettes Smoked (8 hours)	1-hour Data	Concentration ( $\mu\text{g}/\text{m}^3$ ) <sup>b</sup>	Cigarette Smoked (1 hour)
Airport	Mean Day 1 <sup>a</sup> Mean Day 2 <sup>a</sup> 2-Day Mean Range Mean bkgd.	0.61 0.74 0.68 0.48 - 0.99 0.021	261 326 294	Maximum Mean Range Mean bkgd.	1.5 0.72 0.36 - 1.5 0.046	61 75
Junior College <sup>c</sup>	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.035 0.018 0.027 0.013 - 0.044 0.012	30 34 32	Maximum Mean Range Mean bkgd.	0.15 0.051 0.017 - 0.15 <EQL <sup>d</sup>	5 4
Local Government Center <sup>c</sup>	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.066 0.055 0.061 0.042 - 0.073 0.009	59 60 60	Maximum Mean Range Mean bkgd.	0.18 0.097 0.039 - 0.18 <EQL	15 11
Office Complex <sup>c</sup>	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	0.12 0.14 0.13 0.11 - 0.15 0.09	261 251 256	Maximum Mean Range Mean bkgd.	0.28 0.19 0.10 - 0.28 0.06	31 29
Amusement Park	Mean Day 1 Mean Day 2 2-Day Mean Range Mean bkgd.	2.6 2.8 2.7 2.4 - 3.1 0.12	653 719 686	Maximum Mean Range Mean bkgd.	4.6 2.4 0.66 - 4.6 0.17	148 91