Findings of the Scientific Review Panel on
THE REPORT ON BENZO[A]PYRENE
as Adopted at the Panel's April 18, 1994 Meeting

Pursuant to the Health and Safety Code section 39661, the Scientific Review Panel (SRP) has reviewed the report Identification of Benzo[a]pyrene as a Toxic Air Contaminant by the staffs of the Air Resources Board (ARB or Board) and the Office of Environmental Health Hazard Assessment (OEHHA) on the public exposure to, and health effects of, benzo[a]pyrene (BaP). The SRP also reviewed the public comments received on this report. Based on their review the SRP makes the following findings pursuant to Health and Safety Code section 39661:

1. BaP is within the group of chemicals known as particulate polycyclic organic matter (POM) which is listed as a federal hazardous air pollutant and, therefore, was identified as a toxic air contaminant by the Board on April 8, 1993.

2. Based on epidemiological evidence, the International Agency for Research on Cancer (IARC) has classified certain complex mixtures such as soots, mineral oils, shale-oils, and coal tars which contain BaP in Group 1 - carcinogenic to humans. The United States Environmental Protection Agency (U.S. EPA) classified BaP as a possible human carcinogen (Group B2) on the basis of sufficient evidence in animals and no adequate data in humans. The IARC classified BaP as a "probable human carcinogen" (Group 2A) based on sufficient evidence in animals and inadequate evidence in humans.

3. BaP is a product of incomplete combustion; it is present on the surface of respirable, submicron size particles. Its major sources in California are open burning of vegetative materials, mobile sources, rubber tire wear, and residential combustion of wood. The sources of ambient outdoor BaP are estimated to emit approximately 8-13 tons per

year. Other sources include coal combustion and residual oil combustion.

4. Based on air monitoring data collected by the ARB, the statewide population-weighted ambient exposure is estimated to be 0.53 nanograms per cubic meter. The mean near source BaP concentrations in residential areas where wood and vegetative materials are burned during the winter months were 2-17 times higher than the annual statewide population-weighted estimate.

5. Before the introduction of the catalytic converter, mobile sources with spark ignition and diesel engines were the major contributor of BaP emissions. After 1974, in the United States, vehicles were manufactured and operated with catalytic converters. As the pre-1974 vehicles grow older and are taken out of service, BaP emissions will decrease as the number of older vehicles decrease. Reductions in BaP emissions are also expected as a result of decreases in reactive organic gases (ROG) and respirable POM from the introduction of transitional low emission vehicles (TLEV), low emission vehicles (LEV), and "clean" fuels. Respirable combustion products (soot) from non-controlled spark-ignition and diesel engines continue to be a significant source of BaP emissions.

6. There are two dominant removal processes for BaP: a) physical loss processes for the particles on which BaP resides, and b) adsorbed phase reactions of BaP on the particles. The lifetime of BaP due to particle dry deposition is expected to be about 10 days. The lifetime of BaP due to chemical reactions and/or photolysis is on the order of a few hours in polluted urban atmospheres during the summer months. Relatively little is known of the carcinogenicity, or lack thereof, of reaction products, an area warranting expanded exposure and health effects studies.

7. The major sources of indoor BaP in California are tobacco smoking, woodburning in fireplaces and woodstoves, and infiltration of polluted outdoor air into homes. Tobacco smoking indoors raises BaP concentrations by the greatest magnitude compared to other indoor sources. Average concentrations of 2.2 ng/m^3 have been reported in smoker's homes, compared to a level of 0.83 ng/m^3 in a "no source" home. Preliminary data indicates that BaP can accumulate and persist in house dust. For example, investigators reported a range of 1.5 to 41 ppm BaP in an eight-home study in Ohio with an average concentration of 9.6 ppm.

8. In addition to inhalation of BaP, exposure can also occur from dermal absorption and the ingestion of water and food. The levels of BaP measured in various foods range from 0.1 to 60 micrograms per kilogram wet weight. There are insufficient data to estimate the dose of BaP through drinking water exposure in California.

9. Epidemiological evidence for human cancer from exposure to polycyclic aromatic hydrocarbons (PAHs) in complex mixtures is found in studies of roofers, tar distillers, and coke oven workers. Furthermore, human exposure to PAHs such as those found in mainstream cigarette smoke, environmental tobacco smoke, soots and diesel exhaust, may also increase cancer risk. Certain complex mixtures such as soots, tars and oils are in the IARC Group 1 (sufficient evidence for carcinogenicity in humans) based on epidemiological studies. However, in these studies BaP was only one component of a mixture of PAHs and therefore a precise exposure assessment was not made. Consequently, BaP risks are based on animal studies.

10. Exposure to BaP alone results in animal carcinogenicity including cancers of the respiratory tract, gastrointestinal tract, and skin. BaP has the ability through its metabolites to arylate DNA, cause gene mutations in both prokaryotic and eukaryotic cells, induce sister chromatid exchanges in mammalian cells, and produce unscheduled DNA synthesis in mammalian cells.

11. Based on available studies on BaP animal carcinogenicity the range of unit risk for continuous exposure to BaP over a 70 year lifetime is estimated to be from 1.1×10^{-3} to 3.3×10^{-3} per microgram per cubic meter. The "best" upper 95 percent confidence limit value for unit cancer risk is 1.1×10^{-3} per microgram per cubic meter, and is based on the strongest inhalation study reporting respiratory tract tumors in hamsters.

12. The estimates of risk include several sources of uncertainty, including the choice of mathematical model used to estimate risk, extent of absorption of BaP by various routes, variability of response to BaP in different species, the choice of the animal-to-human scaling factors, the choice of the extrapolation model, and the large range of extrapolation (five orders of magnitude) from the BaP concentrations used in the animal experiments to current ambient levels.

13. Based on the "best" value for potential unit cancer risk of 1.1×10^{-3} per microgram per cubic meter and the mean annual statewide population-weighted exposure of 0.53 nanograms per cubic meter, there could be 0.6 potential cancer cases per million over a 70 year lifetime. Based on a population of 30 million California residents, such an exposure could result in a cancer burden of 17 potential cancer cases. Risks to individuals around "hot spots" are substantially higher.

14. Using the OEHHA's "best" estimate of risk, and average indoor concentrations in California homes, exposure to BaP in smoking environments is estimated to result in 0.5 to 2.4 potential cancer cases per million people exposed for most of their day.

15. Table 1 compares the "best" value of upper bound unit cancer risk for BaP with those of other compounds reviewed by the SRP. These 95 percent upper-bound lifetime risk estimates are health-protective estimates; the actual risk may be much lower.

16. BaP is the most studied PAH. However, many other PAHs present with BaP in complex mixtures, such as tobacco smoke and diesel exhaust, are also potential carcinogens. In order to address the carcinogenicity of PAHs in ambient air as a class, potency equivalency factors (PEFs) for some PAHs relative to BaP have been developed using carcinogenesis studies in experimental animals. Table 2 lists 24 additional PAHs and PAH derivatives, their IARC classifications, and their PEFs.

17. Based on the ambient concentrations and PEFs for benzo[b]fluoranthene, benzo[k]fluoranthene, indeno[1,2,3-c,d]pyrene, and dibenz[a,h]anthracene, the combined risk from exposure to these four PAHs is approximately one-third that of BaP.

18. The report also includes inhalation unit risks for four PAHs (dibenz[a,h]anthracene, 3.9×10^{-4} per microgram per cubic meter; 7,12-dimethylbenzanthracene, 2.4×10^{-2} per microgram per cubic meter; 3-methylcholanthrene, 2.1×10^{-3} per microgram per cubic meter; 5-nitroacenaphthene, 1.1×10^{-5} per microgram per cubic meter. These unit risks were developed from "expedited" risk assessments for implementing Proposition 65. It is assumed that the unit risks for inhalation have the same relative activities as cancer potencies for oral intake. Table 3 lists the comparison of unit risks and the risk per million for BaP and these four PAHs.

19. Based on available scientific information, a threshold could not be identified.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on April 18, 1994.

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