

**Seventh Meeting of the Health Effects Advisory Committee (HEAC) for Permissible
Exposure Limits for Airborne Contaminants in the Workplace
California Code of Regulations, Title 8, Section 5155
June 5, 2018
Elihu Harris State Building, 1515 Clay Street, Oakland, California**

HEAC Members Present

Eric N. Brown, Dr. PH, CIH, CSP, The Aerospace Corporation, El Segundo, CA (Industrial Hygiene)
Michael N. Cooper, MS, MPH, CIH, Principal Scientist, Mcooperconsulting LLC, Eagle, ID and UC Davis
(Industrial Hygiene)
Will Forest, MPH, Santa Cruz County Department of Public Health (Epidemiology/Toxicology)
Robert Harrison, MD, MPH, School of Medicine, University of California, San Francisco, CA
(Occupational Medicine)
Mark Stelljes, PhD, SLR International Corp., Martinez, CA (Toxicology) [Attended via phone]
Kent E. Pinkerton, PhD, UC Davis (Pathology/Inhalation Toxicology)
Howard Spielman, Health Sciences Associates and CA Industrial Hygiene Council (Industrial Hygiene)

Public and Interested Parties Present

Dan Leacox, Leacox and Associates
Bob Nocco, Chevron
John Martinelli, Forensic Analytical Consulting
Kashyap Thakore, Toxicologist, California Department of Public Health, HESIS
Michael Geyer, KERNTEC Industries
Robert A. Cary, Sunset Laboratory, Inc., Tigard, OR
Doug Parker, Worksafe
David Ufferfilge, Tesla
Dave Ross, California Department of Transportation
Paul Burnett
Anne Katten, California Rural Legal Assistance Foundation
Lindsay Stovall, American Chemical Council
Jennifer Foreman, Toxicologist, Exxon Mobil
Natalie Rainer, KH Law

Elisa Koski, Cal/OSHA Standards Board
Barbara Weller, California Air Resources Board
Linda Smith, California Air Resources Board

Division of Occupational Safety & Health Personnel Present

Garrett Keating, Chris Kirkham, Eric Berg, and Mike Horowitz

DETAILED MINUTES

Below are detailed notes of the advisory meeting. These notes do not represent a transcript of the meeting, and are simply a summary of the notes taken by the people conducting the meeting. Although every effort has been made to accurately reflect the opinions expressed in the meeting, they should not be considered to be a verbatim record of the proceeding.

Chris Kirkham and Garrett Keating opened the meeting, explaining the agenda and handouts. Keating said that acetates and methyl isobutyl ketone (MIBK) were on the agenda for the morning. He noted that all substances have at least two HEAC reviews; this is MIBK's second.

Butyl Acetates

Summary: The recommendation for n-, iso, and sec-butyl acetate is 50 ppm PEL / 150 ppm STEL and 1 ppm PEL for tert-butyl acetate. Irritation was the basis for n-, iso, and sec-butyl acetate and noncancer and cancer effects for tert-butyl acetate. There was discussion about the exposure route extrapolation used in the EPA health assessment for tert-butyl acetate and the interpretation of rat kidney tumor data in the tert-butyl acetate assessment. Staff agreed to review the extrapolation issue and consider olfactory effects observed for n-butyl acetate. Staff indicated that because of the similarity in their toxicology with the acetate forms, recommendations for n-butanol and tert-butanol would be discussed at the next HEAC meeting.

For the acetates, Keating said the current ACGIH TLVs for "all butyl acetate isomers" (n-, sec-, iso-, and tert-) is 50 ppm for 8 hours and 150 ppm for the STEL [Cal/OSHA's current PEL is 200 ppm for sec- and tert-acetates with no STEL, and 150 ppm for iso- and n-butyl acetates with a STEL of 200 ppm.] Tert-butyl acetate has a more complex toxicology, so we are considering that substance separately. ACGIH grouped the butyl acetates into one TLV based on a similar irritation effect and, upon reconsideration of human exposure data of four hours at 150 ppm leading to minor lung irritation, lowered the TLV to 50 ppm by applying a safety factor of three. ACGIH noted that animal tumor data for tert-butyl acetate may not be relevant to humans. Keating did not see much new literature on acetates and asked Patrick Owens of HEAC to review the ACGIH findings and document for the HEAC summary. Keating plans to further review one study in the draft, a 2001 sub-chronic rat study that shows olfactory damage at 1000 ppm and a NOAEL of 500 ppm. There are no other authoritative bodies that used this study as a basis for an OEL or RfC, although some regulatory agencies (Texas, other) have.

Kent Pinkerton noted that the rat nose is structurally different than the human nose. Also, rats are obligate nose breathers, unlike humans, so the results of exposure will be much different for nose effects.

Bob Nocco agreed--the nasal epithelium of the rodent is more extensive. He noted humans are not obligate nose breathers, but do some nasal breathing so nasal epithelium is a target for exposure.

Keating said similar issues were considered with the rat nose when HEAC looked at H2S. Keating will see if there is comparable PBPK (physiologically based pharmacokinetic) data for the acetate. The ACGIH TLV documents indicate that the acetates are essentially converted to acetic acid and the alcohol, and the acid causes irritation of the respiratory tract. The acetate isomers have different physicochemical properties that could make one more potent than the other. An analytical feasibility section will be added to the butyl acetate drafts (handout); the recommendation was found to be within the feasible

analytical range.

Keating said that the chemicals under HEAC review - benzophenone, SO₂, turpentine and the new chemicals reviewed today would be taken up again at a future HEAC meeting.

Keating moved on to tert-butyl acetate. In response to a question from Mike Cooper, he clarified that toxicology differences notwithstanding, the same TLV and STEL--50 and 150 ppm-- was being proposed by ACGIH for all butyl acetate isomers.

Cooper said he wanted an explanation of the “why” to be included in the document. Keating said for isomers of the butyl acetates, the common endpoint was irritation.

The PEL recommendation for tert-butyl acetate (tBAC) is 1 ppm based on cancer and non-cancer endpoints. A subcommittee of Mark Stelljes, Will Forest and Saeher Muzaffar was convened to discuss the two endpoints. tBAC falls into that class of carcinogens that some attribute to a male rat-specific mechanism considered not relevant to humans-- alpha-2-globulin (A2G). Over the last year, OEHHA and EPA have conducted major reviews of the rat bioassay data for tBAC, arriving at the same recommendation of 1 ppm but for different reasons. OEHHA determined tBAC male rat tumors were not caused by the A2G mechanism and therefore relevant for human hazard assessment. Therefore, OEHHA conducted a standard cancer risk assessment and derived an RfC of 0.8 ppm. EPA acknowledged tBAC did not cause tumors by the A2G mechanism but found there were aspects of the A2G mechanism involved in the tumor response. EPA stated it could not conduct a quantitative cancer risk assessment with evidence that A2G was a factor in the tumor response.

Keating noted that tBAC tumor incidence was derived from oral studies in rats with tert-butanol (tBA), the main metabolite of tBAC. tBAC quickly converts to the alcohol and the chronic toxicity is due to the alcohol. Therefore EPA considered tBA studies relevant to tBAC assessment. EPA also concluded it appropriate to extrapolate from the oral route to the inhalation route. There is little new tBAC literature since the rat study was done in 2007. There are a few studies included in the summary that consider developmental and reproductive effects observed at a higher dose than the kidney effects.

Cooper questioned the validity of a study without evaluation of inhalation effects. Keating acknowledged there was no inhalation pharmacokinetic model, but pointed out a 13 week sub-chronic inhalation study did demonstrate beginning kidney pathologies seen in the 2-year oral tumor study. EPA found it acceptable.

Spielman asked where the tumors occurred in the two-year study. Keating said in the male rat kidney, not the female, which is typically observed for the hypothesized A2G mechanism..

Spielman asked whether the committee was setting a precedent for carcinogenic consideration. He stated he had never been comfortable about how the committee arrived at human carcinogen determinations from a basis of animal studies. He asked how should the committee as supposed health effects experts look at animal data when it only has animal data, a lack of information. He stated if the committee doesn't do it for one material, then why is the committee setting a methodology for how it does it for all chemicals?

Forest answered that the methodology is already there. It's straight-forward. We assume it is a human

carcinogen unless we have evidence to the contrary. It is treated the same way as when you are sure the chemical is a human carcinogen unless there are clear reasons.

Keating commented that the state of the art of risk assessment is to add more pharmacokinetic and dose modeling towards identifying mechanisms in the rat. However, at the end of the day we fall back to the rat bioassay if we don't know what is causing the cancer.

Stelljes said he had the answer for the question about why EPA approved the extrapolation. It says **human availability** in the chronic and subchronic studies would normally also be set aside for dose response analysis. However, overall the NTP 2-year drinking water study was identified as the study most suitable for dose response assessment given its duration, comprehensive reporting of its outcomes, use of multiple species tested, multiple doses tested, and availability of PBPK modeling.

Keating stated that there is a model and that it helps. He would have a look at that for tBAC, not tBA.

Stelljes said that's correct.

Keating said in the rat oral study they did have many more endpoints to benchmark dose model—five different mechanisms and effects from which one was chosen for dose response modeling.

Cooper asked Stelljes if the model adjusted for exposure route. Stelljes said to answer that question he would have to see how they did the modeling, and that information is within another document referenced by this one.

Keating said he would investigate and bring back that information. He said that most PBPK model extrapolations use continuous exposure and run to steady state, which is not always accurate for occupational exposures.

Stelljes said it appears EPA takes the oral data of animal exposure to get a point of departure. Then they modify their extrapolation to the inhalation route by considering blood/air partition coefficient for tert-butanol in the lab animal and humans. That's one extrapolation they used, the blood gas coefficient leading to a ratio of 1.04 that they used.

Keating said EPA was determining a human oral tissue dose. Stelljes agreed and Keating said he would add that to the document.

Keating said that it's relevant that these rat kidney tumors have convincing incidence data and pathology for a subset of chemicals, though some don't. With tBA and tBAC, tumors are found but the pathology is not as consistent.

Forest said it's well established that when you look at large data sets, there is not complete correlation between A2G or chronic nephropathy and cancer. You have A2G cases with and without cancer. You have nephropathy with and without cancer. So just because there is nephropathy and cancer in a subset doesn't mean there is a causal link. You can't demonstrate A2G is a mechanism causing cancer in one chemical—you can't show how it would happen.

Keating asked if the A2G mechanism occurs without tumors; Forest replied affirmatively. You have the

Melnick article from 6, 8 years ago. When the proposal that A2G was the cause of rat kidney tumors and therefore these cancers could be discounted, Melnick did research on a large data set—150 chemicals—finding there wasn't the necessary correlation—there are counter examples to that proposition. When cancer was found, it could not be demonstrated that A2G was the cause. When A2G was found not to be the cause, they moved on to chronic nephropathy and not just the A2G. The initial idea was that microglobulin in rats was the cause of cancer but when that association did not hold up, they moved on to nephropathy. The more recent Melnick article debunks that notion, having already debunked the initial idea.

Keating said he had referenced Melnick paper's assessment of chronic nephropathy in the summary, but would further review it. He noted that very few chemicals get classified as just A2G; there are better criteria that have been developed.

Forest said that these supposedly better criteria are based only on chemicals that increase A2 and cancer. That focus ignores the group with A2G and no cancer and the group with no A2G and cancer.

Keating said an adjustment for occupational exposure will be considered for the next draft. Its fine if there is not consensus on this, but it is appropriate for both mechanisms to stay in the draft.

Cooper said, if they are looking at the human dosages, note that drinking water and shower contributions to dose are not occupational.

Spielman said in terms of setting standards for carcinogens, we are effectively saying a worker can have a 40, 45 year exposure or dose over that timeframe. Our adopting a PEL is essentially setting a dose that will be acceptable over those years. He asked how that concept compares to dose concepts from animal studies where you don't get 40, 45 year studies. We feed the animals huge doses to see if they're going to get some effect. We use NOAELs and the like and work backwards to lower numbers. He stated he had always had some concern about how they take what one might be able to figure out is the dose that caused this in the animal versus a PEL representing a working life dose.

Forest said that's what the process of risk assessment is, the way it has been done for 50 years. It is the standard accepted by the EPA, NIOSH, OEHHA and all the standards setting organizations.

Spielman said for a lot of studies you get down to the assessment and at the end of the day it's the best we have. It took a certain dose to cause the animal to have a problem, and he would like the committee to be able to determine that and determine the safe dose.

Keating said that might be possible for high dose effects. But in the absence of knowledge of whether the effect is a linear effect, which is often the case, we are left the precautionary approach. EPA is always looking for pharmacokinetic modeling and mechanistic factors to better inform risk assessment, but it is not always there.

Harrison said he supports the recommendation. He asked that CERS (California Environmental Reporting System) usage data be included in the revised draft. He asked if butyl acetate is used as a solvent, whether the use would lead to a fair amount of skin contact. He asked if there is any data on skin absorption and the possible need for a skin notation.

Spielman said for this kind of PEL setting, it is important to know what the current industrial exposure range is. Stakeholders may have to provide this information for us to make a good feasibility estimate.

Kashyap Thakore said for the acetate, based upon the recent NIOSH policy, a cancer risk of 1 in 10,000 should be considered. What we have calculated here for tBAC is 0.8 (1 in a 1000), which should be ten times lower, 0.08, to reflect this NIOSH policy.

Keating said Thakore was speaking to the official cancer risk for occupational exposure.

Spielman said 1 in a thousand comes out of a courtroom.

Forest said the Supreme Court benzene decision said that a risk of one in a billion could not be considered significant, while a risk of one in a thousand could be considered significant.

Keating said NIOSH RELS (recommended exposure limits) now use 1 in 10,000 for worker cancer risk.

Thakore said that ratio was established in a 2007 NIOSH document.

Cooper said using 1 in 10,000 as a cancer standard would be a change in policy for HEAC.

Keating said that this would be a justified change because the standard was endorsed by an authoritative body. He said, however, recall that the PEL calculations for both cancer and non-cancer endpoints for tBAC come to the same number.

Spielman noted that OEHHA's Prop 65 cancer exposure guidance is based upon 1 in 100,000 occurrence of cancer.

Stelljes said this ratio may be because Proposition 65 exposure limits are designed for consumers as well as workers whereas PELs only address worker exposure. Spielman agreed the OEHHA number was based upon 24/7 exposure for 70 years.

Methyl-Isobutyl Ketone (MIBK)

MIBK

The PEL recommendation for MIBK is 5 ppm PEL/50 ppm STEL. Developmental effects was the basis for the PEL and irritation for the STEL. Staff reported that MIBK was not manufactured in California and was being transferred from bulk containers for use, such as in resins. Members discussed the importance of the STEL for protecting workers and the validity of the study used to support the STEL recommendation. Staff will report back on the potential health effects from the STEL and provide more details of the study used to support the STEL recommendation.

Keating opened the discussion of MIBK. The current PEL and STEL are 50 and 75 ppm; the recommended PEL and STEL are 5 and 50 ppm. Most of the health effects research was done in a 2007 NTP rat study. A 2003 IRIS rat developmental study was chosen as a point of departure. MIBK, like tBAC, binds to A2G and causes kidney toxicity as well as kidney cancer in male rat. Cancer and non-cancer data from the 2007 rat

bioassay are presented in the summary. There are kidney tumors in male rats, not in females. Following the IRIS approach (2003), the NOAEL from the Tyl developmental study was used, and adjusted for uncertainty and occupational exposure, resulting in an RfC of 7.5 ppm. This methodology differs from previous EPA practice where most developmental assessments did not perform duration adjustments based on the premise that developmental effects were more likely to depend on peak exposure concentrations. This results in use of an adjustment factor in the calculation for MIBK. While not explained in detail in the summary, this EPA calculation method is relevant for a growing number of chemicals.

As another estimate for an OEL, Keating said the 2007 rat study using chronic nephropathy was analyzed using standard NOAEL and uncertainty factor approach. The RfC was 0.3 ppm. Using chronic nephropathy as an effect is not common and not utilized by EPA for tBAC because the chemical dose response for the rat nephropathy is problematic. Both approaches are presented, but Keating chose to base the PEL on the developmental model calculations leading to a proposed PEL of 5 ppm.

The rationale for the 50 ppm STEL is explained on page 12 of the summary as resulting from an exposure study of human volunteers for four hours (Hjelm, 1990). The study measured rapidly rising symptoms related to the subjects' increasing exposure over two hours. This study is unique among human volunteer studies in that response are recorded within the first 30 minutes of exposure. At 50 ppm there are potential effects. Three of eight subjects experienced nose/throat irritation. This observation is based on only this study.

Harrison said the way exposures often occur is intermittent; continuous occupational exposures are rare. So it is probably more likely that someone is going to pay more attention to the STEL because they are using the chemical for a short duration task. He asked if there are data relevant to the setting of STELS; here we are discussing developmental effects at the proposed PEL level. He asked if there are any data showing use of this kind of solvent for repeated short 10 minute exposures with 40 ppm peaks.

Horowitz said it was conceivable, as MIBK is often used as a solvent in resins and similar applications. He stated that the questions he has about it is that as a resin component it is reacting, so he wonders how much of the MIBK solvent is being off-gassed. Theoretically someone could use such a resin containing several solvents for 8 hours repeatedly. He stated he expects most of the time MIBK is in a resin sitting on a shelf that is used only occasionally for an hour and then not used for six months.

Horowitz said he had called some of the users on the CERS list for MIBK. He was finding that it is not produced here; it comes in barrels. In some places they might mix it. He didn't know how they mix it, such as if it is a closed process or not. In many cases some of the larger handlers have not performed any real exposure risk assessment of this chemical.

Harrison said he was really asking if there were reproductive effects from exposure to intermittent peaks near the proposed STEL. Keating said he was unaware of any.

Spielman said at least one STEL, for asbestos, was for a 30 minute period. Historically, one reason STELS evolved as a result of initially high PELs or TLVs such as 250 ppm, and because of ceiling limits that were established but analytical methods at the time required longer sampling times—you couldn't measure the ceiling concentrations but you could measure concentration over a 15 minute time period. So the STELS evolved as a result of limitations to analytical sensitivity. Even ceiling limits became STELS. In terms

of effects on individuals, if one is exposed to a STEL for multiple times, you will exceed the 8-hour TWA.

Harrison said he brought this up because the committee is proposing to set a PEL based upon reproductive effects, but the STEL based upon acute symptoms. He was looking for any guidance in the technical literature for examples of using the STEL for cancer or reproductive effects. In the world of work it is more common for people to be exposed short term

Cooper said he could think of a lot of exceptions for short term limits. He has a slight problem in that the committee is going for a tenfold reduction from the STEL to the PEL. He asked what the committee expects to see coming out of this, and whether it expects a health benefit by lowering the STEL, or is it just the reproductive effects. He asked why the committee had not seen evidence of the health benefit.

Forest answered, it's because nobody looks for it. Reproductive effects are not the kind of effect you can look for from short exposures, unlike wildfire effects which are an acute problem manifesting in a large number of people. With the STEL we are guarding against a level of risk that is small and therefore hard to detect.

Cooper said his question was about the evidence for the tenfold difference between the PEL and STEL.

Keating said the PEL uses the animal model for protection from reproductive effects, and the STEL human data on irritation.

Harrison said he gets calls about solvent exposure risks to pregnant women and he feels the risk is deserving of heightened concern.

Spielman noted that mathematically, if one were exposed at the STEL of 50 ppm for 50 minutes, one would be at the 8-hour PEL.

Kirkham corrected the math, stating it was 55 minutes at the proposed STEL that would lead to a PEL exposure.

Forest noted that exposure at the 15-minute STEL for that long would itself be a violation.

Brown said in the Heljm study at no point was CNS impairment seen and only three of eight reported the irritant effect; they sense an odor. He stated he was unsure that equates to the Cal/OSHA definition of serious impairment.

Keating said there wasn't animal data on this effect, but there were a number of two- and four-hour exposures to humans that do show impairment.

Brown said at the low level there were one or two of eight with irritation. At the exposure concentration of 2.5 ppm there was only one person noting an effect. He wondered if this would be covered by our PEL anyway. He stated the nature of the study doesn't measure impairment as much as it does subjective sensation.

Cooper said the committee is not establishing a NOAEL.

Spielman asked if there was any data on the range of concentrations at which people report symptoms. In the old days, the standard for protection on the predecessor solvent methyl ethyl ketone was set by irritation and the PEL was lowered due to irritation.

Brown said if a chemical has a bad odor, even without health effects, you would record it as irritation.

Keating said the existing STEL was set as a result of CNS effects – “dizziness, headache. Now we have moved to consider the distinction between CNS effects and irritation.

Doug Parker said the summary of the Hjelm study states that at 25 or 50 ppm three of the eight subjects reported nose and throat irritation and two reported headache and vertigo.

Brown said the only time three subjects experienced vertigo was at the mixed exposure (25 ppm plus toluene).

Parker stated the summary says at 25 and 50 ppm. He commented there wouldn't be a question that vertigo would be an impairment.

Brown, referring again to the Hjelm study, said one control experienced vertigo—one of eight people. It only goes up at the 2.5 ppm level to two people at 2.5 ppm and 3 at 50 ppm. The control group was exposed at 2.5 ppm.

Keating said better detail of this study is needed to understand it. He said he would try to improve the summary's clarity. Dave Ross asked when a PEL was set, was the related PPE also determined? Brown answered that it depends upon the exposure and is the employer's responsibility.

Forest said there are some standards that state in detail what you have to do to protect employees, while other standards just state you have to protect them.

The room was queried for comments on MIBK feasibility, and no comments were received.

Keating next discussed the supplementary information handout, projecting the table from the handout on the screen at the front of the room. He stated that the STELs and PELs for the alcohols were listed, but people should recall that the acetates convert to the alcohols when absorbed. For example, a PEL of 50 ppm is proposed for n-butyl acetate which completely converts to n-butyl alcohol. There is a lot of PBPK information on this conversion. At the next meeting he hope to have a revised Priority 1 list to include the alcohols.

Forest said you might find the TLVs and STELs are based upon the irritant effect.

Geyer asked if n-butyl acetate always converts to n-butyl alcohol, or does it convert to a different isomer as well.

Keating wasn't sure but said he would look for that in the literature.

Lunch Break 11:45 AM-12:45 PM

Wildfire Smoke

Kirkham brought the meeting back to order after lunch. We are going to talk about three new substances: wildfire smoke, diesel exhaust and phthalates. We will start with a slide presentation from California Air Resources Board (CARB) representatives Barbara Weller and Linda Smith.

Barbara Weller said she was a pulmonary pathologist and toxicologist and she had studied the health effects of pollutants her entire career.

Slide 3: Weller explained the most devastating and well-established effect of PM 2.5 is premature death from cardio-pulmonary disease in the elderly which has been established as causal. PM 2.5 also causes an increase in hospital admissions for respiratory and cardiac disease. It causes increased emergency room visits for asthma and a number of effects on children including reduced lung function. The groundbreaking Children's Health Study found an increased risk of bronchitis and chronic cough in children. Pregnancy effects associated with PM include low birth weight and pre-term birth. There are some new studies starting to look at birth defects associated with PM such as cardiac abnormalities.

Weller then discussed the most at risk. Older adults with undiagnosed conditions or heart or lung disease are at risk. Children breathe more air per body mass, and breathe more rapidly than adults. Children play outside, and their immune systems are still developing, increasing the risk to children.

Weller said, estimating the effects of PM in California, there are over 7,000 premature deaths, about 2,000 hospitalizations for cardiac and respiratory effects and over 5,000 emergency room visits.

Weller said there are federal and state ambient air quality standards. The National Ambient Air Quality Standards (NAAQS) are the definition of "acceptable" air quality. The California Ambient Air Quality Standards (CAAQS) are the definition of "clean air." These standards are not down to zero risk. There are penalties for not attaining the NAAQS.

Slide 8: lists the differences between the national and California standards for the PM10 and PM2.5. PM2.5 for wood smoke is mostly the fine particles but there are larger particles as well. So wildfire smoke goes all through the size fractions. Wildfire smoke can be a major contributor to total PM2.5.

Harrison asked about the time-frame for these PM air quality readings. Weller said they were 24 hour averages. The scientific literature is always under review, but at the present time, there is insufficient literature to establish short term effects and regulatory levels. The current PM standards are set to protect the general population, not more susceptible subpopulations or occupational groups.

Linda Smith clarified that the standards are designed to protect the more vulnerable groups in the general population, but not the extremely sensitive.

Geyer asked about location of monitoring devices. Weller acknowledged pollution levels from area to area could vary. The next slide shows where the pollution is located, if not where the monitors are located. You can see large portions of California, including the Central Valley, South Coast and Bay area, are out of attainment for the California standard for PM2.5, and wildfire smoke contributes to these levels. Based on this concern, Weller said, USEPA along with CARB, CDC and US Forestry Service released

a document to provide local officials with guidance for wildfire events. Slide 12 lists the guidance for public officials on various PM_{2.5} concentrations from “good” to “hazardous.” Closing schools is something to be done only for a very high exposure. But anyone with a pre-existing conditions should take extra steps during a wildfire event.

Geyer suggested that when there is a wildfire with high Santa Ana winds, the amount of dust being blown might give a false reading. Weller said the concentration of dust would be far below the concentration of smoke. Dust has different health effects than wildfire smoke, and there are places in California like the Salton Sea where the mineral content of the soil makes windblown dust a particular health concern. But, in general, windblown dust is not nearly as toxic as wildfire smoke.

Harrison asked where an occupational standard recommendation could fit in. He stated that he knows the AAQS were not developed for occupational exposure, and asked what the presenters thought. And whether addressing exposures in outdoor workplaces during a wildfire would be reasonable.

Weller agreed the AAQS were not developed for occupational exposure. Kirkham said the current PEL is for Particulates Not Otherwise Regulated (PNOR) for respirable particulates is 5 mg/m³, while total PNOR is 10 mg/m³. Weller noted that the PEL for PNOR was far higher than the ambient PM_{2.5} standard, but also, wildfire fighters were a healthier subgroup.

Spielman noted that the AAQS were for a 24-hour exposure, while occupational exposure is evaluated as a 40-hour exposure.

Weller addressed the next slide, which examines the CARB study of the wood burning ban in the San Joaquin Valley air basin. They wanted to look at the effect on hospitalizations and the effect on PM for the three years before and after the regulation went into effect. They looked at adults and elderly 45-64. After the wood burning ban they got an overall decrease of PM_{2.5} of 12 percent. There was a decrease in the coarse particles and in the fine PM. They looked at urban and rural areas, finding a little better effect in rural areas. They looked at cardiovascular disease hospitalizations, ischemic heart disease and chronic obstructive pulmonary disease (COPD). There were not many COPD cases, but for the 65 and older age group, there was a significant reduction of hospitalizations for cardiovascular disease with the wood burning ban of seven percent in the San Joaquin basin, and a little more effect in rural areas. Ischemic heart disease (heart attack), 16 percent reduction.

Spielman asked if the study included the effect of formaldehyde, often a by-product of wood burning. Weller said the study didn't have any speciation. Weller then, in response to a question, clarified how to interpret the slides about the study. These are cases of hospitalization, not PM readings, she said. They estimated 200 reduced cases of cardiovascular disease, 101 fewer cases of ischemic heart disease in rural regions.

On the next slide about California wildfire-related health studies, Weller pointed out the epidemiologic studies of the 2003 and 2007 southern California wildfires. Many studies show increased respiratory hospital admissions, especially for asthma for the very young and the elderly. One study did not find the increase in children-asthma hospital admissions, but it did find an increase in rescue medication, fast acting inhaler, for the condition. Another study looked at the pregnancy outcomes of women pregnant during the 2003 wildfire, finding an increased incidence of low birth rate. A study at UC Davis Primate Center, at the time of the Trinity and Humboldt fires of 2008, examined early adolescent rhesus

monkeys found immune and lung deficits that persist into maturity. The next slide describes the follow-up study they are doing to see if this cohort of affected monkeys can pass the deficient functions from mother to child, sort of an epigenetic effect. Results of this study are due next year. This concludes the slide show.

David Ufferfilge complemented the presentation and CARB's website with the Air Quality Index and associated recommendations, which Tesla provides to its employees.

Keating asked if there were studies of spikes in PM_{2.5} that were not wood smoke. Weller said there were not as many studies looking at short-term exposures; most look at chronic exposures. CARB has studied the short-term effects of exposures from traffic, and there are historic studies of N. Carolina highway troopers' exposures to traffic. Lung function deficits have been found in such studies. A lot of the insults to the lungs are similar to those seen with wood smoke. The jury is still out as to whether such effects are more respiratory or more cardiovascular. CARB found a cardiovascular effect with overall PM levels. One thing making it difficult to study wood smoke is the fact that our monitors are stationary. So for a several-week tire fire near Fresno, they only caught the plume from that fire once or twice due to shifting winds. Meteorology has an effect; huge fires create their own meteorology.

Keating said the tire fire was a good example of the type of fire that could ideally be used to compare effects with those of wild fires. Since CARB samplers are stationary, has CARB ever used information that could integrate PM data over more area, such as satellite data?

Linda Smith said CARB is looking at that right now. They are looking at four kilometer grids of the state of California which they intend to improve to single kilometer views. There is money in the budget to refine this satellite information, which of course is only good during daylight.

Horowitz asked if the stationary monitors were passive or battery operated, direct reading or filter-based, and what were the analytical methods used? Weller said CARB had everything—all of the above, depending on the pollutant. Most monitors were at stations with lighting and electricity, like a little building or room, where they are long term.

Cooper asked if co-pollutants such as carbon monoxide were pulled from the PM_{2.5} data. Weller said for this study CARB only looked at PM_{2.5} during the wood burning season. The only factor that changed in the six years of this study was the wood burning ban.

Kirkham asked if there was particular information that went into the specific CARB recommendations for the six different PM_{2.5} ranges. Weller said there are references in the guidance document.

Harrison said his similar question was if there was data on PM_{2.5} levels during major fires. Is there a database of PM_{2.5} levels for the recent North Bay fires?

Weller couldn't say about a database, but remembered that for one community levels of 300 micrograms of PM_{2.5} were reported.

John Martinelli echoed the plaudits for the usefulness of the Air Quality Index. During the fires people were told that filtered air in the hospital was not very good, but better than anything they would experience outside in the community. Trying to come up with occupational standard, translating from

the 24 hour recommendations, is difficult. He compared the difficulty to trying to determine how much water someone was exposed to when underwater in the ocean. Studies of indoor wood smoke exposure give valuable air quality data. But, with a wild fire there are a lot of additional toxins from structures burning, you've got trailer parks burning. Many other things besides wood—even grass—are burning in a wildfire.

Weller agreed there is a tremendous amount of difference between a brush fire and a structural fire. There are all kinds of toxins in a structural fire. She said she was not familiar with studies of structural firefighter exposures. Studies of wildfire firefighters show lung function decrements during the season, with recovery after the season. You have to think of this as injury repair. Of course, firefighters are very fit; they are not the sensitive population of asthmatics, children and the elderly, but they are getting a high dose.

Linda Smith said she didn't think CARB had done research on health impacts of structural fires.

Dave Ross asked about baselines. Weller said the best they could do was the wildfire guidance documents.

Anne Katten said CARB and community groups were very active in supplying N95 respirators to farmworkers during the fires. Farmworkers were harvesting grapes during the North Bay fires, and harvesting strawberries in the Ventura fires. Cal/OSHA did institute the advisory requiring the wearing of N95s during the Ventura fire, which was a very much needed first step in developing a final standard. Since this is seasonal work, agricultural workers will work if the jobs are offered. A lot of the agricultural work is piece rate, so for farmworkers there is a high respiratory rate. So CRLA very much supports the development of a standard for wildfire smoke.

Pinkerton noted, as alluded to, that PM2.5 from wildfires is not the same as PM2.5 from ambient conditions, and that's one of the critical things to look at. Even though we have a PM standard we use for air quality that may not fit for wildfires. Wildfire PM might be quite more toxic than ambient.

Paul Burnett said there are water tender truck drivers who deliver water to the fire lines. He cited research, *Review of the Health Effects of Wildland Smoke on Wildland Firefighters and the Public*.¹

Weller asked if this research focused on short bursts of high exposure or longer duration.

Pinkerton said both types of exposure are important. The duration that the smoke lingers and its transport are important factors. The Central Valley is a perfect example of fires occurring to the north followed by transport of the smoke to the southernmost portions of the valley.

Spielman said he didn't see this committee or anyone setting a PEL for this kind of exposure. A potential approach used for other standards, is if we have some sense of what major exposures might be, people could return to remediated locations. If we have some sense of the range of exposures, we could put together a work practice standard that would be protective. You don't need numbers to have a work practice forbidding walking into a cloud of smoke. The best you could expect from an occupational point

¹ Inhal Toxicol. 2016;28(3):95-139. doi: 10.3109/08958378.2016.1145771.
https://www.fs.fed.us/pnw/pubs/journals/pnw_2016_adetona001.pdf

of view is a best practice standard. You can't control the PM levels in a wild fire. Think of the problems caused by a volcanic eruption.

Harrison said he supports the development of a standard of some type. Collect available data from the ARB. I think this will wind up with personal protective equipment for wildland firefighters that's feasible for them. Promote and develop respiratory protective measures NIOSH has been working on, NFPA has worked on, and what Jimmy Johnson worked on at Lawrence Livermore.

Cooper said there has to be adequate measurements as well. Harrison agreed.

Summary: Wildfire smoke, diesel exhaust and phthalates were for discussion by HEAC; no health summaries or PEL recommendations were presented for these substances by staff. Speakers from CARB presented background on wildfire smoke and Staff presented information on diesel exhaust and the phthalates. A comprehensive discussion of these topics followed and Staff agreed to address comments in a follow-up report.

Diesel Exhaust

Keating said that the diesel topic would be another open discussion. There is no recommendation or health summary today. There is some data in the literature, with this handout. Diesel was discussed last meeting as a candidate for the Priority 1 list for the coming HEAC year. Diesel has been listed as a human carcinogen by several authoritative bodies. Human epidemiology is available for the cancer risk assessment, though no agency has done the quantitative risk analysis. NIOSH and NCI co-authored this meta-analysis showing the excess cancer risk for exposures of 25, 10 and 1 micrograms per cubic meter and excess cancer risk per ten thousand, the NIOSH cancer metric. Particulate matter, elemental carbon is a surrogate for diesel PM, old diesel, and new diesel—there is a lot of terminology. This was a meta-analysis of three cohorts—two truck driver studies and an underground miner study. Underground mine studies with exposures in the 100's of micrograms was the initial basis for risk assessments.

There are non-cancer effects, Keating said, but interpretation is affected by the existence of new technology and old technology. CARB may want to weigh in, but I think in 2007, new standards were set for diesel fuel's particulate and other pollutant constituents like NO₂. When we at HEAC look at studies we will have to confront which form of diesel was being used, and what endpoint.

Linda Smith said the difference between the new technology and old technology is how much gets out, but the exhaust is the same. Elemental carbon is the measurement used.

Robert Cary said if you look at diesel exhaust, 80 per cent is elemental carbon. Elemental carbon therefore is used in certain locations as a surrogate for the entirety of diesel exhaust though we know there are adsorbed organic hydrocarbon particles on the elemental carbon. Our people multiply the elemental carbon concentration by 1.2 or 1.4 to get total diesel particulate.

Geyer noted ammonia is added these days to some diesel engines. Cary said the ammonia helps oxidize the carbon.

A speaker said ammonia addition may lower particulate emission but makes what is emitted more toxic. Linda Smith agreed the new technology dilutes the exhaust but potentially different toxicity makes the situation difficult to interpret.

Keating asked Smith if she was familiar with the emissions study by the HEI that looked at the old and new diesel. Keating said he was not familiar with it, and Smith said she was only a little bit familiar. Keating said it seemed to show different levels; but on a per gram basis it is just as toxic. He discussed a 2009 NIOSH table showing levels for different above-ground activities. Mean concentrations vary from single digits to 30 or 40 or more micrograms per cubic meter. Some mechanic jobs had very high levels. He stated that he was not asserting this is the current level of diesel, rather he was presenting this information for the sake of this discussion. NIOSH has published broad scale measurement of the range of exposures seen in the workplace. I suspect many of the entries on the table are pre-2007. We have below-ground mining data, with most above ground data from the transportation sector. There are some firefighter studies.

Keating then mentioned a more recent study of a fracking operation showing exposures of 10 micrograms per cubic meter. Note this study utilized personal sampling, and not area sampling. The numbers are close—10 versus 16 or 17 micrograms per cubic meter. There is no question diesel is carcinogenic notwithstanding some re-evaluation of potential confounders and efforts to firm up the true value of the cancer risk estimate. The cancer risk value would be a big driver of a PEL, so interested committee members would have to do more work on this estimate.

Cooper asked if this would be a HEAC or separate committee. Keating said diesel has previously been listed as a special committee substance, but he hadn't really thought about it. Diesel information would take some time to compile; it wouldn't be ready in three months. DOSH could try to set up a separate committee, but a subcommittee with a few members of HEAC might be sufficient with Keating compiling the information and circulating it. We've done that with tBAC, for example, with Will and Mark.

Eric Berg said MSHA has an elemental carbon standard for diesel exhaust. The federal register final rule for this MSHA standard has a very detailed analysis which we can go back to for studies and risk assessment information.

Kirkham said he wasn't sure the MSHA elemental carbon standard has come out; there has been some pushback. There are problems with elemental carbon in coal mines, but these would only affect application of the rule to coal mines. They've currently have a diesel particulate standard of 160 micrograms total carbon per cubic meter, which was reduced from a previous limit of 400 micrograms total carbon per cubic meter.

Berg agreed that the elemental carbon MSHA standard was not due to be implemented until 2022, but the risk assessment has already been published and therefore is useable.

Geyer said the sampling assumes there is no other carbon source upwind. For example, he has sampled up wind of sugar mills and found interference. You either assume there is no upwind cofounder or you have to separate the two carbon sources by sampling and analytical methods.

Keating answered a question by Spielman that the elemental carbon standard of MSHA was meant to

apply to non-metal mining. Spielman said silica rock could be a confounder. Keating said that is part of the re-evaluations of the cancer risk assessment going on. Part of the problem is the human epidemiology was based upon years of exposure, but lacked personal sampling data which could be utilized for standard dose reconstruction.

Spielman said this uncertainty about sampling would be one obstacle for us to deal with. Keating said it is in our policy to develop a health-based proposal, with feasibility assessment coming afterwards.

Harrison said there is enough in the literature to support a PEL based on a cancer endpoint.

An unidentified commenter said the background, such as from highway traffic, should be excluded from personal sampling totals. Berg said it all goes in the lungs.

Harrison said he recommended proceeding with a sub-committee that would deal with the technical and feasibility issues like outdoor exposure that have been mentioned. Diesel is a substance that seems amenable to starting with the existing literature and cancer risk assessments.

Brown said we have to define where occupational exposure meets environmental non-occupational outdoor exposure and draw the line between the two. There is a background level and an occupational level. For example, someone commutes on the 405 for four or five hours a day exposed to a lot of diesel—maybe their air conditioner is broken and they keep the windows open, and are exposed to a lot of exhaust. Then you have people who work and do the same thing, like truck drivers.

Cooper said the complexity that Brown and Harrison have articulated show there would be a tremendous number of stakeholders for diesel rulemaking.

Keating asked if there was an answer to Brown's question that diesel is an ambient pollutant.

Kirkham said we have PELs for environmental pollutants such as the carbon dioxide PEL of 5000 ppm and the PELs for carbon monoxide and ozone.

Berg said that he'd heard that the strip in Las Vegas had experienced carbon monoxide levels over 100 ppm, four times the PEL. But OSHA does not subtract this background concentration from personal exposure.

Brown said maybe our job is just to set the occupational levels even if people who are not working, such as residents in a wildfire area, might be overexposed to an occupational standard.

Spielman said the issue becomes larger when an environmental standard comes close to an occupational standard. Generally where workers are exposed to the same risk at work and at home, they are more at risk at work.

Keating said he saw Spielman's point, a lot of PELs were set decades ago. Keating asked how the committee addresses Cooper's question regarding whether this should be an issue for HEAC as a whole. Keating suggested a HEAC subcommittee of HEAC review the epidemiology literature and present findings on cancer as a clear end point to the full committee later.

Kirkham said NIOSH and ACGIH had not really set a recommended exposure limit for diesel particulate. The Australian Institute of Occupational hygienists has suggested 100 micrograms per cubic meter of elemental carbon. Keating said that number was associated with underground mining. Austria and Ottawa, Canada, may have exposure limits.

Berg said MSHA's limit was 160 micrograms total carbon per cubic meter. Keating said CDPH recommends 20 grams per cubic meter. Thakore said HESIS had reached this number using 1998 OEHHA epidemiology findings and the NIOSH cancer risk figure of 1 in 10,000 to derive the 20 microgram result.

Spielman said there is consensus that diesel exhaust is a carcinogen and that people are exposed to it at work. We should go forward and see if we can set a reasonable standard.

Forest agreed with Cooper; the proposal of a specific diesel PEL as a question before the full committee would more than fill this room with all the interested parties.

Keating said again he proposed a review of epidemiology on this question. Would that bring in a lot of stakeholders?

Forest said absolutely, but that doesn't mean we shouldn't do it.

Cooper said the idea that an employer has to do something about an exposure he has no control over on the ambient side would be controversial.

Forest said with wildland smoke you are not necessarily sure of what you should regulate, but with diesel you have reasonable data on elemental carbon so there is a discrete substance that falls within the purview of this committee. As opposed to wildland smoke, which I don't think we can take on.

Brown said he thought the committee could take on wildfire smoke.

Forest said if there was a good meta-review of wildfire smoke that identifies components, you perhaps could. Diesel has a single parameter, elemental carbon, that we can reasonably use on which to base a standard.

Geyer said elemental carbon is produced by any high temperature burning. Forest fires produce tremendous amounts of elemental carbon. The difference is wildfire smoke seems to be 90 percent organics and maybe 8 to 10 percent elemental carbon. Elemental carbon is not necessarily an indicator of diesel exhaust, as regular gas engines also produce it. I don't know if NIOSH fully understands that. The NIOSH approach works in a mine but you can't apply it where there may be other environmental sources of elemental carbon.

Smith mentioned the 2012 IARC study which Keating said he would take a look at again. Smith mentioned carbon dioxide as another component of diesel potentially contributing to health effects, as was NO₂. Truckers were upset when CARB initially told them emissions had to be reduced. CARB recommendations took a decade to finalize and implement the new diesel emission rules.

Parker said occupational diesel exposure does not only occur outside. In warehouses forklifts and trucks create indoor exposure.

Kirkham asked the CARB representatives about diesel engine and fuel technology changes in the late 2000's. When you stated gram for gram the emissions that come from a new diesel engine were the same as the old, that was in the engine context. In the fuel context, is the exhaust that comes from the new fuel equally potent?

Smith said the sulfur content of the new fuel was less, therefore sulfur dioxide in the exhaust is a reduced component of toxicity. However CARB is not able to say overall toxicity is reduced as the same organic toxins are produced by the new fuel burning. (PAH's, Carey interjected, are always produced when burning any fuel.) In an experiment with the new fuel that CARB did with primates, the organics were filtered out, but toxicity was still observed.

Keating said a purpose of today's meeting was to figure out for the three substances we are talking about, which ones do we want to put on the Priority 1 list. From a planning perspective, diesel is a good candidate for P1, but we will revisit that. So let's now discuss phthalates.

Smith said CARB has an indoor air quality section that is very interested in phthalates.

Phthalates

Keating said phthalates are on the Priority Special Substances list because of controversy, dynamic changes of use in the work place, and the cumulative risk methods proposed for phthalates. Because of the common endocrine effects, the recommendation of the National Academy of Sciences (NAS) and other agencies is to do a cumulative risk assessment for this class of chemicals. The question for the committee is should Cal/OSHA address the four regulated phthalates with PELs at 5 mg/m³. Those limits are not based on the endocrine effects.

Keating showed some slides to stimulate the discussion.² The first slide showed the number of phthalates in percentages of people it was found. He showed NHANES data from 2005--14. DEHP declined, DINP on the increase for the 95th percentile. DEHP has some of the most prominent health effects, so other phthalates are replacing it in products. The NHANES results are not occupational exposures. Mostly the exposure comes from cosmetics, food wraps, plastic toys and bottles, and many other sources. To do an assessment of cumulative risk, you do a hazard index, which is a quotient with the exposure over the reference. If the reference value is 10 mg/m³ and you are exposed at 1 mg, the index is 0.1. If you are exposed to two or three phthalates, then those quotient add up. If the total is greater than one, you have exceeded the reference concentration for the combined hazard. The reference concentration is combined from the reference concentrations for each individual phthalate. This is what the NAS and others are recommending, although it is not as simple as described. Not all phthalates have reference values from EPA, so in some phthalate assessments European reference values are used.

Keating said different phthalates have different potencies for different endpoints. One method to account for this is called "relative potency." To do a cumulative index for a specific endpoint, phthalates are scaled to the most toxic one, the one that has the lowest reference value. Keating stated he did that here [shows slide]. DBP has the lowest benchmark dose, so it is the most potent. Other phthalates are

² <https://www.dir.ca.gov/dosh/DoshReg/5155-Meetings/Phthalate-handout.pdf>

scaled to that and their reference value for that endpoint lowered accordingly.

Another factor in phthalate hazard assessment is how often these cumulative exposures occur. NHANES has urine samples from 2000+ individuals but how many of those samples have multiple phthalates in them? e. This slide is from the most recent NHANES data set. So under frequency of phthalate mixture in the 95th percentile—the high exposed group, most don't have a mixture. Only 123 individuals in that group have two phthalates, a unique subset of this population where most people have some exposure, but not at the 95th percentile. Alternatively, you could look for multiple phthalate exposures above the 50th percentile for which a cumulative hazard quotient could be developed. For 2000 subjects, only 21 had a hazard index greater than 1. Of those, the most common pairing was DINP and DEHP. The data displayed here is mostly based on environmental, consumer, exposure. For occupational exposures, since there is a background of environmental exposure to phthalates, researchers look at the increase of phthalate in urine between pre- and post-shift. They try to determine what tasks in the workplace may be contributing to the total phthalate exposure. Keating referenced the slide of data from a NIOSH study of PVC and rubber manufacturers.

Cooper asked if this data was relevant to California. Keating said there was very little CERS data on phthalate use in California. PVC manufacturing and PVC film production are the two most commonly found. DBP and DEHP are the two most toxic phthalates. DINP, is less toxic, and is replacing DEHP and other phthalates.

Harrison asked about any studies of exposures besides PVC production. Keating said there are nail salon studies. A study of cosmetic counter perfume workers measured phthalate levels pre- and post-shift. Air concentrations were measured at 1 to 2 micrograms per cubic meter.

Harrison said it would be useful to figure out the NAICS code of PVC worker studies. From the code he might be able to track down how many workers might be similarly exposed, as he has access to some employment data bases. He is just trying to figure out what is the extent of occupational exposure in California.

Stelljes said he wasn't sure we should add phthalates to the Priority 1 list unless we were sure of similar modes of action for the different phthalates.

Keating agreed that was a key point for developing cumulative risk assessments for differing phthalates.

Jennifer Foreman said on one slide with the bar graph some grouped phthalates were using reproductive effects, but others used different effects. They were incorrectly put together.

Keating stated, in agreement, phthalates need to be grouped by toxic effect for proper cumulative assessment. He circulated Foreman's handout to the group.

Spielman asked if inhalation was the only route of exposure. Keating said no, it's absorbed dermally, its aerosolized. They are not particularly volatile compounds. But to Cooper and Harrison's point about California exposure, in most cases the phthalates are coming in pre-produced products like PVC pellets or floor tiles manufactured elsewhere. The phthalates are added during the manufacturing stage. OEHHA has safe harbor levels for floor installers placing phthalate-containing pliable vinyl tiles where there may be dermal or hand to mouth contact as well as inhalation exposure. The medical device industry also uses phthalates. This industry may have some manufacturing in California, but it will take

more effort to find out who is using phthalates in California.

Harrison asked if there was a sampling method. Keating said Tenax tubes were the recommended sampling media. If you pull a sample, possibly you get multiple phthalates for which a cumulative hazard assessment would be appropriate. But yes, there are established methods.

Keating said we need to upgrade our PELs for phthalates. Currently the PELs are inconsistent with the current reference values. Most OEL values around the world also remain at 5 milligrams per cubic meter. The only updated value he found was in Germany where they are proposing 0.5 milligrams per cubic meter for DEP. Sara Janssen, who is not here today, is very interested in this. She agreed with looking at PELs first and then at cumulative exposure and how to incorporate that into a PEL.

Keating said there are multiple endpoints so this will take a fair amount of toxicological review. There might be a way to have two PELs, one based on the endpoint of a single phthalate, the other based on common endpoints for several phthalates via a hazard index. Two PELs for a single chemical, with different endpoints. He had not presented on this today, but some phthalates have endocrine effects, some reproductive and developmental. There is no formal hazard index yet from OSHA.

Foreman said the cumulative risk approach may not be necessary, given the typical ratios products tend to have for the different phthalates. For example, in the production of rubber gaskets, if you apply the NAS approach, which is the maximum cumulative risk approach, if you have a value greater than two you might have a cumulative risk. A value smaller than two indicates a single value will suffice. If you look at geometric means for the maximum daily exposures you see a range generally from 1.1 to 1.3, so most of the occupational exposure is being driven by a single value.

Spielman said in Southern California, most PVC use in manufacturing is in the form of pellets, which are fed to extruders. He did not recall raw material manufacturing, which is where you would see most phthalate exposure.

Keating asked about thin PVC film manufacturing industry. Spielman and Foreman said the issue is the making of raw PVC. Keating speculated that the medical industry might do raw PVC manufacturing.

Foreman said 94% of occupational exposure to phthalates is from DEHP.

Harrison asked Keating if the issue was how to proceed with the phthalates – as P1, P2 or other?.

Keating said yes the phthalates could be placed on P1 or P2 or back on special chemical list. He summarized that for the P1 list we have SO₂, benzophenone, turpentine, butyl acetates, and depending on advice today, wood smoke and diesel and/or phthalates. These latter three are complex; Keating would have to prepare several reports for a proposed PEL to be presented. There are epidemiology questions that need review. The others are simple PELs that are straight forward. Keating isn't sure all three of these should go in at once. Wood smoke and diesel fit the HEAC conventional pattern more than phthalates for which the methods are more in flux. We know a lot about diesel and wood smoke in California but not much about phthalates.

Spielman asked if the endpoints or guides for diesel were as severe as for other substances. Keating said no. Spielman said more workers may be exposed to diesel. Keating said his goal for the next meeting

was to finalize the P1 list, perhaps adding two of the three substances discussed today. Between now and then the Division will discuss the issues internally and will try to find out more about phthalate usage in California. Spielman suggested CIHC could help.

Cooper said diesel might be a yes, but a specialty committee. Kirkham asked about the glutaraldehyde special committee experience. Cooper and Forest said it had been a separate committee lasting a couple of years assembled around the workplace exposure issues of that chemical. It was successful, and it was a focused committee; there was a lot of involvement, separate from HEAC. Keating said we won't discuss that today, and Cooper said it was a resource issue, as would be a separate committee for diesel exhaust. Spielman said diesel was a different animal, with more known about it, so it may be more amenable to being handled effectively in a separate committee.

Keating said it sounds like HEAC wants to consider diesel, it's just a question of whether we can do it. The Division may have to decide if it is feasible to approach diesel now in this way. If it is going to be that big of a stakeholder event, it could slow down other things the Division is trying to accomplish. So we will take the issue under advisement.

Berg asked whether the committee wanted to think about wood smoke or PM2.5 in general as a separate committee.

Spielman said his preference would be for a work practice standard.

Berg asked how would PM2.5 be any different than PM10 for which we already have a PEL of 5000. We have tons of data on PM2.5. A standard for just smoke may not be as protective but if we make a PM2.5 standard, just based upon size, at least we would have something. It would be more protective than we are at now. The PM10s PEL is based upon "not otherwise regulated"; we are ten times higher than the most dangerous particle size category for PM10.

Spielman said the emphasis on wildfire smoke seems to be just based on particle size. Toxicology might highlight another aspect.

Berg said, yes, size is the criterion for outside the immediate area. He asked whether the committee should we try to go for smoke, or do we try to set a general standard for PM2.5. Different options.

Harrison recommended focusing on wildfire smoke, which would focus on wild land firefighters and other occupations exposed to wildfire smoke. They are not like their structural fire counterparts who have self-contained breathing apparatus and an upgraded approach to health and safety programs. From his perspective, in terms of having an impact on a very concrete group in ten years, if there is a wildfire smoke standard—or even just discussion of a standard—a focus on wild fire smoke will be more effective than a PEL for PM2.5.

Pinkerton said he believed wildfire smoke was the larger issue, and we will in the future have more and more such fires. It's going to be a growing issue.

Harrison said wildfire smoke is hazardous. Even if OSHA only has the discussion about addressing wild land smoke, it will be a step forward. If you talk to wild land firefighters as I have, who are out there on the fire lines, this is part of the job. Wild land smoke is not identified as a particular hazard. I don't think

the hazard is realized as an OSHA regulated hazard.

Horowitz said wild land firefighters may continue to be exposed at their rest locations. Harrison said hot shot firefighters tell him the air is better in rest areas. Harrison said he thinks wild fire smoke is a very worthwhile issue to take on.

Brown said wild land fire fighters are far more impacted than vulnerable groups like the elderly. Berg asked about farmworkers. Brown said they would not be as impacted as firefighters. Brown also argued that diesel and phthalates had larger occupational exposure profiles—more workers affected--than smoke. We should prioritize and maximize our effort, he said. Wildfires are “glamorous,” and dangerous, but the affected population is small. Brown asked who the occupationally exposed population to PM 2.5 includes, and suggested firefighters

Spielman favored the concept of a work practice standard and expressed concern at applying a PEL. Berg said NFPA has a respirator for wild land firefighters. Spielman said maybe CARB could help us estimate the exposures that can be expected for the various scenarios. That information could be used to set a work practice standard. As said earlier, maybe wearing respirators is not feasible, so involving stakeholders in the discussion is important; the suppliers of protective equipment could talk about what they can provide. SCBA respirators would be impractical. Berg repeated, NFPA does have a standard for wild land fire respirators. We just need manufacturers to come up to it.

Cooper said you would get less resistance to a work practice standard for wild land firefighting than to a PEL for wood smoke. Not just a small group is exposed; logistical support is also exposed. However, a work practice approach will generate less resistance than putting a number to it.

Keating noted the ranked exposure levels of CARB that drive certain actions. You could have work practices, but by pegging it to a number, you would have a trigger for initiating practices. Keating asked about how one would make those work practices happen.

Cooper asked about what one would look for, such as smoke opacity. In his opinion, setting a number will result in more digging in of heels.

Forest said fine particulates are so clearly very important. He thought to the committee was in agreement that this should be a work practice standard but he was not sure this committee is the place to develop it. It might merit a subcommittee.

Cooper said you have fine particulates and an array of chemicals that is complex.

Berg said we could use fine particulates as a surrogate. We could spin off the smoke as a separate thing.

Cooper said PM2.5 is too broad to get compliance.

Forest said most studies of small particulates he is aware of were early ones that do not look at specific toxicities of the fine particles. Like asbestos, the physical form appears to be the problem. While it's correct that there is a chemical contribution, for fine particulate, per say, it is the form.

Carey said EPA has given up at looking at most specific chemical composition of fine particles except for

sulfate concentration of small particles in aerosols. Complex chemical reactions in wood smoke particles are problematic, not just the size and shape.

Harrison said he saw a PM2.5 standard as very broad and potentially more superficial as opposed to a specific occupational standard for wild land firefighting, which could be more comprehensive and perhaps more impactful.

Berg said that the committee is discussing both, one a separate committee that could discuss a work practice standard for wild land firefighters and two, another this group could work on ultrafine particulate matter.

Keating asked whether the ultrafine group would address chemicals, not the particles, per se.

Berg said, no, just the particle, total PM2.5.

Carey noted the long history of generation of PM2.5 particles by combustion, engines, diesels. Air quality standards were developed to address particles from those processes. Kirkham agreed. Carbon particles were more harmful constituents than the nitrogen based combustion products.

Berg asked Carey if total carbon was a better measurement than elemental carbon used by MSHA. Carey said he thought it would make a better standard. Berg mentioned Parker had served at MSHA during the time it was developing its air standard of 160 micrograms of carbon for mines. Parker said that was developed earlier. Carey said there is a multiplication factor of 1.2 to account for the predicted organic components of the engine exhaust.

Keating said it was time to wrap up. He stated that the committee needs to reshape its position on wood smoke particles, because he had heard different ideas from different committee members; he was not sure there was a consensus.

Forest said there was a lot of support for a work practices approach. Keating agreed, but asked if the committee is considering the 2.5 standard.

Cooper said diesel and wood smoke were a little intensive for this committee.

Keating said the question underlying all of this was, how do we address these special substances? He stated DOSH will take the advice of the committee. We will come back with a Priority one list probably without the phthalates on it and not much else; we haven't added other chemicals. Maybe the phthalates, if we can characterize California usage better.

Cooper asked about CDPH-recommended chemicals. Keating said HESIS had some recommended chemicals which he would review again. We can probably fill out the list with three or four chemicals. So that's what we will do. We will need to figure out work practices for wildfire smoke, how to address diesel—what kind of committee format.

Berg said yes, separate committees for these two.

Keating said he will put out for the committee consideration some chemicals some members have

recommended. For butyl acetates, he is going to look into the olfactory endpoint. With tert-butyl acetate he will look at the PBPK analysis and how that might inform the 1 ppm number. The committee didn't talk about the alcohols today; he is going to elevate n- and tert-butanol to the P1 list. For the acetates we have analytical feasibility but we still are looking at economic factors to add to those reviews.

Parker said the risk of nail salon workers to the acetates should be addressed in the coming year's discussion of these chemicals.

Keating stated that we will look at the irritation effect of acetates. The only reason alcohols were on the agenda today was whether or not to add them to the P1 list. We did that before lunch when we reviewed the table with the alcohols and acetates.

The date for the next meeting is September 4th, the Tuesday after Labor Day. The Western Steel Council will be giving a presentation on manganese at that meeting. Butyl acetates likely will be on the agenda, as well as more chemicals for the P1 list.