Fifth 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 December 12, 2017 Elihu Harris State Building 1515 Clay Street Oakland, California

HEAC Members present

Eric N. Brown, DrPH, CIH, CSP, The Aerospace Corporation, El Segundo, CA (Industrial Hygiene)

Michael N. Cooper, MS, MPH, CIH, Principal Scientist, Mcooperconsulting LLC, Eagle, ID (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)

Sarah Janssen, MD, PhD, MPH, Occupational Medicine Department, Kaiser Permanente, San Francisco, CA (Occupational Medicine)

Patrick Owens, MSPH, CIH, Shell Oil Martinez Refinery, Martinez, CA (Industrial Hygiene)

Mark Stelljes, PhD, SLR International Corp., Martinez, CA (Toxicology)

James Unmack, CIH, Unmack Corp., San Pedro, CA (Industrial Hygiene)

Michael Bates, PhD, UC Berkeley (Toxicology)

Kent E. Pinkerton, MD, UC Davis (Epidemiology)

Howard Spielman, Health Sciences Associates and CA Industrial Hygiene Council

Public and Interested Parties

Erica Stewart, Kaiser Permanente
Dan Leacox, Leacox and Associates
Bob Nocco, Chevron
Bill Taylor, PASMA
Saeher Muzaffar, California Department of Public Health, HESIS
Richard Warburton, ChemDAQ, Inc.
Matt Spencer, US Poultry & Egg Association
Bruce Wick, CalPASC
Lindsay Stovall, American Chemistry Council
Kashyap Thakore, Toxicologist, California Department of Public Health, HESIS
Tom Jacob, Chemical Industry Council
Russel Johnson, Associated Building Contractors

Russ McCrary, CA Ironworkers Employee Council
Mara Ortenburger, Worksafe
Bob Brown, Bay Area WSPA
Rob Neenan, CA League of Food Producers
Emma Wilson, CDPR
Steve Derman, Medishare Environmental Health and Safety Services
Tracey Davies
William Cyd, Health Scientist, Cardno ChemRisk
Scott Dotson, Managing Health Scientist, Cardno ChemRisk
Stewart Holm, Forestry Council
David Woodward, East Bay MUD
Mindy Nelson
Calvin C. Willhite, Risk Sciences International
Hank McDermott, HJ McDermott, Inc.
Marti Fisher, California Chamber of Commerce
Mitch Steiger, CA Labor Federation
Courtney Mizutani, Mizutani Environmental
Craig Conlon, MD, Kaiser Permanente
Larry Medina, Draeger, Inc.
Elizabeth Treanor, Phylmar Regulatory Roundtable
Tom Davies, The Herrick Corporation

David Kernazitskas, Cal/OSHA Standards Board Staff

Division of Occupational Safety & Health

Garrett Keating, Steve Smith, Eric Berg, Kevin Graulich and Mike Horowitz

Steve Smith opened the meeting, introducing the Division personnel present, pointed out the sign-in sheets and handouts at the rear of the room, including recent stakeholder information not yet on the web page but which had been previously provided to HEAC expert committee members. Only the cover letter of the very large recently received aluminum comment was on the back table; it will be posted on the web site in full soon. The expert members present introduced themselves.

Smith stated the agenda provided for discussion of hydrogen sulfide (H2S), manganese and peracetic acid (PAA) in the morning and, after the break, 2-butoxyethylethanol (2BE) and 2-butoxyethylacetate (2BEA), methyl isobutyl ketone, and finally a recap of the recommendations we heard from the last meeting on aluminum. Finally, if we are not all worn out, we will discuss concepts on how to prioritize the next set of substances for 2018.

Steve announced his retirement at the end of the month after more than 30 years with the Division.

Hydrogen Sulfide

Garrett Keating made two major changes to the HEAC H2S document since the last meeting. He expanded the section on lactate analysis based upon the Bhambhani data , digging into the relevance of this subclinical effect. He extrapolated the increase of lactic acid from 15 and 30 minute intervals to 8 hours work period.

On the table constructed from this data, note that test 1, 2 and 3 are different. These were tests on 16 male subjects. Crudely put, test 1 is the most rigorous condition; the least healthy subjects exercised to fatigue. VO_2 max oxygen carrying capacity of the subjects is in column 1 at 41.5% average for those subjects. Tests 2 and 3 are less rigorous tests with healthier subjects put on exercise bikes at half-maximum power with subjects VO_2 max of approx. 50%, a healthier condition. Though this shows variability, in general you see lactate buildup in all three tests, with significance in test 1. In test 1, subjects start out with slow, easy exertion (V1) and then ramp up to V2 and V3. At all exercise conditions, significant lactate buildup is seen at 5 parts per million (ppm) H2S. Test 2 on healthier subjects they did not see the same significance as test 1. Test 3 raised the concentration to 10 ppm and achieved significance.

Keating addressed relevance of this to safety by using linear extrapolation to extend the graph to longer time periods. At 30 minutes blood lactate max is at 8, at 60 minutes 16. Lactate elevation beyond average blood lactate of 4 is considered lactate buildup. The second addition to the H2S document examined an olfactory risk based estimate for an OEL. An existing analysis of human olfactory H2S buildup was adjusted for occupational exposure. Schroeder, the paper author, uses a very complex human PBPK [physiologically based pharmacokinetic] model to predict a No Observed Adverse Effect Level [NOAEL] of 5.0ppm. Some stakeholders have critiqued the Bhambhani study because it was not used by the National Academy of Sciences [NAS] for an Acute Exposure Guideline Level [AEGL] assessment. Two NAS committees have referenced Bhambhani for AEGL development; one committee discounted the study, the other used it as a NOAEL.

Mark Stelljes asked if the extrapolated rise of lactate levels with time reflects physiological reality. Is linear rise of lactate levels during longer exercise in general in the literature? How much rise to be toxic?

Keating said there is a lot of data on very short-term lactate rise from *high exertion* activity, such as sports or firefighters with the rise actually non-linear. Above the lactate threshold, less oxygen is available for use, so it sort of builds on itself. No studies found for the scenario I extrapolated: four, or eight hour, *moderate* level exercise. Some guidance for recommended VO_2 max levels for activities like firefighting and lumber work indirectly address this scenario. Considering how H2S works, Bhambhani is interesting and creative. My extrapolation is not validated by any other study.

Kent Pinkerton asked if blood lactate was a measure of H2S exposure. Keating said since blood acts as a filter for H2S, blood lactate levels might capture individual variability. Blood lactate of four is the standard average threshold for lactate levels to affect physiology. In an H2S atmosphere, it is more a measure of the extent of continuing exposure, as lactate levels rise .

Cooper said H2S detoxification is related to rising levels of lactate and cytochrome oxidase. Various modes of respiration provide pathways for toxicity buildup. Basing a PEL on a subclinical measure of lactate when lactate increases normally with exercise concerns me. I do not know the lactate level increases during moderate exercise as in Bhambhani versus lactate changes during a recovery experiment. As you exercise more you respire more, which is another mechanism to help detoxify some of the H2S coming in; some may not get to the blood. I am more in favor of taking a clinical effect like eye irritation as opposed to looking at the lactate linear extrapolation, which I'm not sure we have a basis for doing. Does using a subclinical effect that can't be seen by a physician lead to a health effect?

Patrick Owens asked if Bhambhani measured oxygen deficiency or neurological function. Keating said the study focused on subclinical changeover to lactate metabolism.

In response to Michael Bates, Keating explained that each subject served as his or her own control. The subjects went through exertion cycling at four H2S concentrations: 0, 2, .05 and 5. Only at 5 ppm do you see a difference in test 1. The 0 ppm test served as baseline; this was a good way to reduce variability.Keating told Stelljes that going from low to high H2S concentrations did not bias the Bhambhani tests because the subjects breathe steady consistent doses through face masks.

Bates asked if there was any acknowledged level of toxicity in the Bhambhani study. Keating said that in studies about health the lactate threshold of 4 is utilized for that purpose. There were no health effects observed in the Bhambhani studies.

Howard Spielman said H2S is an old toxin with exposed populations in various industries. Is there any data from those populations that indicate a need to bring the level down to this proposed level? The issue of material impairment needs to be addressed somehow. In all the studies we looked at, only the study on the pulp workers looked at actual exposed populations.

Robert Harrison said the definition of material impairment is not restricted to human epidemiologic or clinical studies, but it also includes animal toxicology and other data. So an effect on lactic acid metabolism or olfactory effects is a material impairment.

Smith said most acute exposure studies can't be done on people or in the workplace. For example, the chemical concentration affecting ability to exit a confined space. The amount of data on human chronic low levels of exposure may be less than ideal, so animal studies are relied upon.

Spielman said Russian colleagues trained animals to negotiate a maze and then exposed the animals to increasing concentrations of solvents until they could not figure a way out. This behavioral change was considered a material impairment, with the mouse or rat data translated to the potential for human change. In this country we never did that, relying instead on clinical data for deriving our OELs. Somewhere between the two approaches is something we could agree to call "material impairment," but I don't know where that line is.

Cooper said the question is whether Bhambhani is looking at an incremental lactate increase with respect to the H2S concentration beyond lactate increase from exercise. Is that a material issue that resolves when the exercise stops? Mode of action of detoxification of the H2S is uncertain. Would the lactate increase be material from a clinician's perspective? Not to me from a laymen's perspective.

Sarah Janssen was unfamiliar with the normal bell curve for blood lactate level increase during exercise. We measure lactate levels to see if there were changes from what it was, at baseline. How the Bhambhani levels correlate with normal clinical levels, I don't know off the top of my head.

Cooper said in a hospital blood lactate is measured when patients are exercising. Janssen said they may have changes in their metabolism. Keating said he thought basal blood lactate level is not 4, but is between 0.5 and 1.5. The Bhambhani test was at 4 and above.

Dan Leacox said the term "material impairment" comes out of the statute that authorizes the state to set PELs [quotes from CA Labor Code 144.6]. There has to be a path to a material impairment or loss of functional capacity to an employee demonstrated. Often animal studies do make that point. Eric Brown said the LC also states the PEL chosen must be the one that "*most adequately assures*" no employee will suffer, so it's more about potential expression of that endpoint. Leacox agreed the LC 144.6 included that but he was trying to emphasize that "material impairment" was a construct conferring authority to protect employees.

Calvyn Willhite served on the ACGIH TLV committee and later the NAS AEGL and submarine committees. For the H2S AEGL 3, there is a lactic acid buildup endpoint health effect considered adverse. Is the lactic acid buildup seen in Bhambhani a genuine adverse effect? Look at the studies and endpoint used by the NAS submarine committee and compare and contrast it to Bhambhani. This will reduce the need for extrapolations and you can then frame your reference. A caveat from the H2S AEGL committee discussion, is don't set your number so low that when you consider Yellowstone National Park, the Geysers, or other locations with high H2S odors that it makes you look foolish.

Keating said the AEGL submarine committee used eye irritation as the health endpoint while the other committee used the asthmatic effect but they cite the Bhambhani study as a NOAEL, as one of seven studies cited. Both of them use extrapolation. Keating said he had relied upon extrapolation because he had been unable to find data on low H2S exposures during moderate exercise.

Owens said Bhambhani did not address H2S eye exposure. Brown said the debate was whether, if extended to eight hours, the Bhambhani exposures would lead to additional fatigue and, whether that increase would be clinically significant. If we as a group conclude this happens, then it becomes a material impairment.

Cooper said for exercise sustained over time, you would see physiological changes based upon the exercise alone; I don't know where those lines [exercise effect vs H2S effect] crisscross. At low H2S levels, they cross somewhere, but I don't want to hang our hat on the linear extrapolation to 8 or 12 hours. I'd be more interested in a clinical effect for which a physician can say, for example, "You've got eye irritation." I think if you took the eye studies, even the most conservative ones, you hit a value of 10 ppm and divide that by 3 for human variability. You get 3ish, which to me is about 5 ppm. Or 1. But you eliminate a lot of issues like feasibility mentioned by Mr. Willhite.

Stelljes said people would not work or exercise 8 consecutive hours. There would be rest breaks and lunch. Brown agreed, adding that there would be additional factors like heat that would reduce the period of exercise, for example, as in construction activity.

Stewart Holm said pulp mills have changed a lot. Most H2S exposure is at the digester. There are probably four or five people on the floor, but they are in operating booths watching levels of sodium hydroxide and sodium sulfide and adding wood chips. In the wood yard workers are operating machinery. The level of exertion in pulp mills is not like that of lumberjacks.

Keating noted that other industries like refineries and agriculture do demand a high exertion level.

Stelljes said he agreed with Cooper that if we went back to the eye irritation endpoint we would eliminate many of these issues.

Owens said that in the summary of the recommendation, in middle of the first paragraph, where the exposure duration is extended to 8 hours, what are the concentrations?. Keating said 5.

Harrison said H2S is a very toxic gas from a clinical perspective. High acute exposures interfere with cellular metabolism. To me an increase in lactic acid reflects a cellular mitochondrial cytochrome oxidase dysfunction or impairment. That is the clinical significance of the Bhambhani study. A deductive case. This dysfunction is an early marker of material impairment relative to a substance that with high short term exposure is lethal, and from an occupational medicine point of view I would prefer to be health protective to prevent that from happening. It is a material impairment, a measurable effect on cytochrome oxidase at 5 ppm. The second question was about the Brenneman study which shows damage to the rodent nasal passages at 30 ppm. The current PEL is 10 ppm. Can you comment on this study?

Keating said material impairment often has to be demonstrated via extrapolation from animal studies. Brenneman was a rat study with 80, 30, 10, 0 ppm H2S. This subchronic study found nasal lesions at 80 and 30 but none at 10. This is the basis for the IRIS/HESIS 2000 parts per billion [ppb] value for humans shown in the table. Others have used modeling of the differences between rodent and human nasal passages along with PBPK modeling to estimate the H2S concentration that would cause similar effects--21 ppb for a NOAEL continuous exposure, but needs scaling for occupational exposure.

Owens said the benchmark dose, not NOAEL. Keating agreed: it was the 5% benchmark, a good way to go. Fundamentally, the animal data is used to estimate a NOAEL. In the Schroeder paper it is 5 ppm, but I scaled it to 2.5 ppm, considering the relevant adjustment factors.

Russel Johnson said CA Associated Building Contractors, Central California Chapter has a training and workforce development center in Bakersfield for 70% of California oil and gas production. We trained over 50,000 employees for this industry in the last five years. Workforce safety is highly emphasized in this industry, but lowering the PEL to 1 ppm will require mitigations detracting from safety. For example, fans would have to be utilized, increasing tripping hazards. Respirators would be necessary, impacting the older workforce which may not pass the fit test, pushing this skilled and highly trained group out. At 10 ppm they stop work, very safe for the oil and gas industry. Anyone in the field can execute a stop work order if they perceive conditions are not safe. Our members drill wells and deal with H2S on a regular basis. A PEL of 1 ppm is not in the best interests of the workers.

Leacox said the oil and gas industry uses direct reading instruments for H2S detection. A plume can cause H2S concentration to rise rapidly so below 10 ppm is not feasible for a set point for stopping work. While it is difficult to accumulate feasibility information, many folks have showed up today. Assessing feasibility of a proposed PEL is part of HEAC's mission, quote: "*Discussion with interested parties on the*

feasibility and the impact of the proposed new or revised PEL"

Keating said upon the basis of the discussion today, he will have to bring back a new health effects assessment, so holding back the feasibility discussion until then would be appropriate. There will be additional opportunities to raise the feasibility issues. The discussion indicates members think the summary document analysis should not be the basis of a PEL. Stelljes agreed, stating the amount of research on the endpoint proposed was not sufficient to base a revised PEL on. Keating said in his review he will focus on the eye effect; there are a number of ACGIH references on eye effects, and he will also bring back the olfactory PBPK analysis.

Cooper addressed Harrison on cytochrome oxidase, looking at page 10, test number 1 wasn't looked at, for number 2 and 3 the males actually went down at 5 ppm. We are not seeing an increase in cytochrome oxidase reduction along with increased H2S concentration. In my view I still need something clinical to hang my hat on. This isn't very conclusive, it doesn't look like cytochrome oxidase is the mechanism for detoxifying H2S in this study.

Spielman noted H2S has a built-in olfactory avoidance mechanism, although there is the olfactory fatigue issue. But it has a built-in warning factor. Secondly, if we set an STEL of 10, and walk away from it, does that do the job?

Stelljes said a STEL might almost be more appropriate. Will Forest agreed, it's sort of the decision of the last meeting. Focus on killing people. A good STEL and Ceiling would address the chronic exposure issues. Exposure may vary, but lower chronic exposures will result from this approach.

Brown was not sure. Some industries are prone to plumes, but other industries working the same process are going to have that consistent long term 8 hour, 12 hour exposure. Plumes may not address long term 8 hour exposures. Stelljes said he's not sure the long term low exposures produce a material impairment.

Cooper said there is an advantage to the STEL/Ceiling idea is that is a lot of industries will take the PEL and set an Action Limit at ½ the PEL. A PEL of 5 would result in an Action Limit of 2.5, which could be measured. What I am having trouble with though is that at the lowest of levels there may be a feasibility/reliability of measurement issue. So perhaps the STEL idea is a way to get around that.

Keating said he will come back on work on eye effects and look at the STEL idea, which he is skeptical could substitute for a PEL. Stelljes said you don't have to do away with the PEL.

Holmes wanted to address the summary's mention of the Fiedler (1990) study's mention of anxiety symptoms resulting from H2S exposures. Shows a slide of a graph from an ANSI presentation on this study illustrating that statistical and clinical biological evidence is lacking for the anxiety effect. You will note that at 0.05 ppm, 0.5 and 5 ppm anxiety symptoms go up slightly at 5. Severity of three on a scale of 100 is seen as important. ANSI notes the 0 ppm is an error. So I don't think this study supports anxiety symptoms as being material impairment.

Harrison agreed that the Fiedler study was not adequate to support anxiety as an appropriate health end point. I'm much more concerned about the effect on cellular metabolism. And I don't think Keating was putting a lot of weight on the Fiedler study, he was just reporting on it.

Holmes said the Yarmount study on asthmatics was important for him, since it was of his industry. I've submitted extensive written comments to show this study has a lack of applicability for this process. Note some lung function tests were lower but standard lung function test differences were not statistically significant and the study does not show systematic lung function decline.

Manganese

Keating said the recommendation is for 0.01 mg/m^3 total dust. We used the Roels study, using both NOAEL and benchmark dose (BMD) approaches. No major changes made to the draft; I am opening up for any more questions about factors. An Uncertainty Factor of 3 was added for potential developmental, pre-natal effects. Linda Morse agreed the literature on developmental effects was not strong enough to be the basis of a health-based PEL. Other Uncertainty Factors were considered for effects on welders, particularly for women welders in the third trimester of pregnancies who experience a spike in manganese during that phase. As mentioned in the draft it is difficult to base a PEL on this effect for now, but is something that needs watching. I probably need to revise that estimated Uncertainty Factor.

Cooper said Roels study levels weren't based on human controls. As Hank McDermott's written comment notes, Roels was based upon post exposure assessment, not pre-exposure. That's my concern with Roels. Keating said Roels was a matched controls study of smelter workers.

Spielman said the Roels results were probably pretty good because for smelter workers sampling results were not plagued with the uncertainties of many studies of welders that don't specify whether the sampling was performed with the cassette located inside the welding hood/face shield or outside of it on the lapel, and as the welder lifts or lowers their shield. This uncertainty makes quality of data on welding often pretty spotty. From my own experience, sampling location (in or outside) with regard to the welding hood can result in a factor of difference of two or three times. So with welding, what is the quality of data when we look at sampling data when comparing with medical findings?

Brown said welding sampling varies hugely. Exposure variability will average out over time notwithstanding the many sources of variability in addition to sampling methodology—for example, by sample size and type of welding. Cooper said a lot of bad data doesn't make good data. Owens noted that use of the geometric mean had recently been questioned.

Keating said there were many well-documented welding studies internationally, and many NIOSH studies and publications. Roels was used because it was consider the best data set.

Pinkerton asked about the particle size in the Roels study. Keating said particles of respirable size with a

cut off below 5 [microns].

On feasibility, Russell McCray of the ironworkers said there were costs associated with the proposed PEL-- huge for 100,000 square foot shops, to move the air around. Besides welders, the fitters, inspectors and others working nearby are potentially affected. In the field, a welder in 110° Palm Springs, today is exposed at the current PEL. A lower PEL requires this worker to wear a respirator. Mobility requirements may make supplied air respirators impractical. A separate advisory committee should be convened to consider the impact of a lowered PEL on this industry with its 100,000 employees directly welding and 30,000 accessory workers alongside. Welding consumable substitution is not always practical given job specs. Sometimes new consumables create worse exposure; when we adopted 3-11 nickel wire, it looked like volcanoes going off.

Johnson said the building contractors' association would like more time to comment on H2S. The same feasibility issues with respirators and masks for H2S protection in the oil and gas industry are pertinent for many welders in the construction industry.

Keating said a large amount of comments had arrived over the weekend which the IH side had not had a chance to evaluate. From my perspective on the science and health effects of welding, the bioavailability of manganese in welding fume versus other forms of manganese is infinitely better. Blood levels in welders are higher from shorter exposure times than for other manganese operations. The Cumulative Exposure Index (BEI) data indicate that welders have smaller cumulative CEI's but have noticeable effects. Without downplaying feasibility, this tells me the health effects are something we really need to look at. And this is human data. With new MRI imaging techniques for Mn, much more exposure data on Mn is becoming available.

Brown said that for workers welding outside in Palm Springs, the wind should reduce exposures well-below the proposed PEL. Inside or enclosed spaces should, as a rule, have fixed ventilation systems like fume hoods which should eliminate the need for respiratory protection. We have a known clinically proven long term health effect that requires change in work practice or engineering control.

Harrison said ACGIH in 2012 was at 0.02 while the recommendation here is at 0.01. Is there a real difference between these two numbers? I agree we ought to gather more information about engineering controls and feasibility. Have companies made a change in response to the ACGIH TLV since 2012?

Hank McDermott said there is anecdotal data that outdoor welding on carbon steel will require a respirator, and as we move up to the specialty steels, we would be over the TLV. Industry is in a transition. Consumable manufacturers are trying to reduce the amount of manganese in products. In regard to the current TLV, there is concern about arc welding on carbon steel outdoors. McDermott had no published data on hand, and Owens thought there may have been an article in the industrial hygiene journal by Rappaport, perhaps using OSHA sampling data, documenting welding fume exposures. Owens said in refineries it was common to build containments for welding projects

Brown said fume extraction devices could be successful in pulling away the fume without disturbing the shielding gas. Owens agreed local exhaust was feasible in fixed settings but noted in refineries workers moved along a pipe. Spielman said a small fan blowing laterally across the breathing zone could blow away the fume and protect the worker without disturbing the shielding. Owens said fans could cause problems for workers nearby. Spielman said fans' advantage is not moving thousands of cubic feet per minute of air through an exhaust pipe.

Michael Horowitz said a recent article on reducing chrome VI welding exposure in AIHA's *The Synergist* evaluated substituting base metals, consumables, or welding processes, giving percentages of reduction in metal content in the fume, including for manganese fume.

McDermott said manufacturers' work on reformatting content of consumables was going on independently of anything OSHA was doing. A lot of new toxicology has been done since the studies relied upon by ACGIH in formulating its 2012 manganese PEL. We should look at that, what's relied upon in the draft summary is not the most modern stuff.

Keating agreed there were newer studies, but these newer studies had great exposure data without good health effects data. The effects of 0.02 are well supported. Roels, subsequent studies, Myers and Young. Exposure and effect measurements are not as good in the more recent studies.

Brown said he'd missed anything other than anecdotal evidence that refuted the proposed PEL value. Keating said there hadn't been time to absorb or adequately review the written comments recently submitted, and also he could take another look at newer studies.

Spielman said if newer studies did not indicate anything different than the ACGIH TLV, then 0.02 sounds pretty good.

Harrison supported a reduced PEL based upon the neuro behavior effects of manganese exposure which he has seen clinically in his patients. The articles I read generally agree about this health effect endpoint. The question is dose-response. Unless there is evidence to the contrary, I agree with 0.01 or 0.02—or say 0.01+. I agree with your recommendation. So I think the main issue is feasibility.

Owens said the proposed PEL would mean refinery welders would need Powered Air Purifying Respirators (PAPR) for all routine welding.

Forest strenuously requested people submit comments at least a couple of weeks before the scheduled meeting. I would urge we not accept submittals receive after two weeks prior to the meeting date. Everybody knows the meeting dates, has months to work on it, and we got 15 papers yesterday. That just makes no sense.

<u>Peracetic Acid (PAA)</u>

Keating said no changes were proposed for the PAA health recommendations. Comments were received from ChemDaq which manufactures a device that gives real time exposure data. We've received some information on the analytical method indicating that method is possibly acceptable. Given the rapid increase in usage of PAA, a PEL is warranted even though some stakeholder input from the last meeting did not agree with that conclusion.

Spielman said one health end point was olfactory irritation which suggests a STEL and Ceiling Limit. Regarding the detection and analytic method, the committee will always be faced with advancing technologies. This committee may have to adopt a practice for substances for which chemical analytical methods are not approved or validated by NIOSH or OSHA. In the PAA case it may be worth doing that because if we don't set a limit then we would be holding off protection. I think we shouldn't do that.

Cooper said that one of the comments that came in late on December 11th from the septic and antimicrobial group mentioned a NOAEL of 0.5 and a LOAEL of 1.25 for eye irritation. This would be useful data if it's something we can obtain. I didn't see a reference in the material provided. Can we obtain the data?

Steve Derman said as past chair of an American Industrial Hygiene Association (AIHA) hospital working group, he recently provided NIOSH information on from a survey of the working group's members. The AIHA working group has discussed PAA extensively over the last several years. There are still no verified sampling and analytical methods. Even the devices on the market have not been verified or field tested. We have taken these devices out in the field, but they probably are not reliable at this time. We had no way to know how accurate the levels are. As a regulatory group, there's no way of determining what the exposure levels are. So a PEL is not feasible as we don't have the information. We've surveyed our membership and have some data which we will share with this committee, but the information is insufficient. Employee complaints of irritation prompted our members to attempt to assess the exposures. We used the Hecht method to make assessments but we were unable to determine a cause and effect relationship. I will provide that information as well. Also, a change in the proposal since last time is a skin notation;

Matt Spencer said the poultry industry is doing a comprehensive exposure assessment study testing PAA levels using both conventional industrial hygiene methods and direct reading equipment. We ask the committee to review that data when it is completed in early 2018 .

Rob Neenan said in the absence of very good data or analytical methods and measurement tools to assess exposure, it seems premature to move ahead with rulemaking. I did an informal survey of our members. Some don't use it at all. PAA is used to clean tanks in the winery industry, but I don't think it is being used more than it was in previous years. Keating asked if PAA was being sprayed on with the worker at a distance or was it being hand applied by the worker. Neenan said his impression was it was being used mostly in pipes and tanks, but some were using it as a general purpose cleaning compound.

Spielman has seen PAA used in the dairy industry to clean big tall vats and silos. That's internally sprayed and dropped down. They also may have a pot to sterilize some of their smaller implements. I would ask the people who are using PAA, in the absence of a standard, what are you doing in your own house? Neenan replied that he only had anecdotal information, but basically there are work practices in place. If they have

a sense there is a problem, it is addressed.

Spielman asked what are you doing, what practices have you adopted? Do you rely on the worker themselves to step outside for fresh air when they experience irritation? The point is, we have a material that is increasing in usage and we know it is an irritant. We have these concerns about delaying action to set a standard, but obviously PAA users know there is a problem. Tell us what you are doing about it; that would be helpful.

Richard Warburton said ChemDaq has customers who use their direct reading monitoring equipment to measure PAA levels. Spielman said the big question was what ChemDaq numbers means; show me the data on the development of your sensor that shows your instrument does what you say it does.

Erica Stewart said since the health endpoint we are trying to control for is sensory irritation perhaps a STEL is more appropriate than a PEL. Three points. PAA is used to clean environmental surfaces. Two, as a complex mixture with the also irritating chemicals hydrogen peroxide and acetic acid, how can one say an effect is due to PAA alone. Third, we don't know what peoples' exposures to irritating cleaning chemicals are outside of work (ex – 2-butoxyethanol). Not that we shouldn't establish a PEL, but establishing a safety factor is complicated given these additional exposures and the sampling analytical methods not being validated. Setting a limit too low would leave no alternative for surface cleaning but to switch to bleach, and there is no PEL for hypo chloric acid although we know bleach has problems of its own.

Harrison said I agree we can't set a PEL without a validated method. Does Cal/OSHA think we are going to get a validated method, and if so, in what time frame? We need to do something about PAA. If a method is on the horizon, my recommendation is to do some groundwork but wait. If we are not going to get a method soon, we need a different approach than a PEL. Does anyone know the status of method validation efforts?

In response to the question from Harrison, Horowitz summarized federal OSHA and NIOSH developments on method validation. OSHA created a known concentration and tried to recover their validation criterion of 80% of a chemical's initial concentration, in this case using the Hecht method which uses treated derivatives to separate PAA from hydrogen peroxide. They recovered 70%. You might call this a semi-quantitative method. So there is a method you can use to semi-quantitatively assess exposure. When OSHA used a recently recalibrated or refurbished ChemDaq machine, they were able to get nearly perfect recovery of the starting concentration of PAA. NIOSH will perform a full investigation of PAA, including chemical analysis development, exposure assessments and animal toxicology. The timeframe for that is the NIOSH timeframe. NIOSH took ten years to fully evaluate diacetyl. Do we wait for NIOSH or do we utilize the totality of information we have now?

Steve Smith said the answer to that question is we are not waiting for OSHA or NIOSH. We have a method that has a higher level of uncertainty, but it is a method.

Spielman noted that when NIOSH or OSHA validate a method, it is typically based upon knowledge of what

the PEL or STEL is going to be. They do it not so much for the users, but rather for its utility for the enforcement program. This is not to say these methods cannot be modified—such as collecting more volume in a shorter time span. No OSHA or NIOSH method for a specific 8 hour TLV doesn't mean a method cannot be utilized for the purposes of an evaluation. Cooper, agreeing with Spielman, said people don't want to wait for NIOSH. Is California seeing a similar increase in PAA use as nationally? It sounds like it is. Most industries would rather have some kind of handle on what to look for instead of waiting for a general duties clause enforcement action.

Will Forest said ACGIH usually has a sampling and analytical method in mind when it adopts a TLV, and it has adopted a TLV for PAA. [CORRECTION: ACGIH has only adopted a STEL for PAA].

McDermott said actually the preamble to the TLV booklet states there are cases in which there is none. [He was referencing this sentence from the TLV booklet: "*Similarly although there are usually valid methods to measure workplace exposures at the TLVs and BEIs, there can be instances where such reliable test methods have not yet been validated."*—editor]

Derman noted there are instances of methods that work well in the lab in a controlled environment but don't succeed in the field because of the presence of other chemicals which are confounding factors causing the method to be thrown out. We should make sure that anything we do is appropriate and accurate.

Smith said the recommendation—with a higher level of uncertainty--was for 0.4 STEL, 0.15 PEL. The alternate recommendation was for just going with a STEL.

Harrison said he believed there were circumstances in which PAA is utilized more continuously, such as in the poultry industry. If so, then I think we need both a STEL and PEL. I support the proposed recommendation for both a STEL and PEL. Cooper asked if we could define a Limit of Detection and Limit of Quantification for the STEL and PEL. Smith said there would be a high level of uncertainty. For the PEL people would just have to prove 30% over that. Not the gold standard, but the silver standard…

Cooper said from a statistics perspective we might want to go with one decimal place rather than the two in 0.15—let's make it 0.2.

Smith said we will continue to take in more information from stakeholders, we encourage you to submit it. This is primarily a health-based recommendation but we don't dissuade continuing to give us information. Cooper noted that at least one group provided NOAELs and LOAELs.

Smith said on-going studies will be factored in as well. Liz Treanor said a deadline, such as January 31st should be established for submittal of the information from studies currently being done. Smith noted the Division ultimately submits a formal rulemaking proposal to the Standards Board. However, compiling that rulemaking takes quite a while, so if relevant information is submitted during that time, the Division can still utilize it.

Adjournment for Lunch

Smith summarized the results of the morning session. H2S and manganese would be continued on for the next HEAC meeting. The recommendations for PAA from the discussion for a STEL of 0.4 and a PEL rounded to 0.2 would be incorporated into a revised HEAC summary document for this substance.

2BE and 2BEA

Keating noted the single HEAC draft summary for two substances, 2-butoxyethanol (2BE) and 2-butoxyethylacetate (2BEA). The acetate is quickly absorbed and metabolized to the ethanol, so both substances are metabolized to the same toxic metabolite. All the literature in the summary is on 2BE. At our last meeting I summarized the two main approaches to 2BE toxicology, one by the EPA and one by OEHHA. Each used the same animal study and different health end point. EPA used a hemolytic endpoint while OEHHA used ocular nasal irritation. I have proceeded to develop a recommendation based upon the hemolytic effect, derived primarily from a rodent study with PBPK scaling. The assessment and recommendation is on page 7 of the HEAC document. Though the 1 ppm recommendation is mostly based on a rodent study, one study I cite observed a slight effect on humans in an occupational cohort. I won't go into all of the calculations on page 8, but that was a continuous exposure in that study, so there is some occupational scaling. This is a pretty conservative approach. Rodents are more sensitive to the hemolytic effects of BE than humans—that's cited in the studies of BE incubation with blood samples from rodents and humans. A cancer risk study was specific to the mouse, and under US cancer risk assessment guidelines the study was not considered relevant for human risk assessment. The Biological Exposure Index is discussed in the final paragraph. 2BE is readily absorbed through the skin, so there are multiple routes of exposure for this chemical. There is a skin notation recommendation because of this dermal permeability. ACGIH recommended against a skin notation because they became convinced that the inhalation route was primary. But I cite a 2011 study that demonstrates an increase in workers 2BE BEI results with obvious contributions to the rise from dermal exposure. Dermal absorption of this compound is well known.

Stelljes said he agreed a dermal notation was definitely necessary. Secondly, I question whether we need a inter-species uncertainty factor of 10, given the point of departure is a more sensitive species (the mouse) and because it is a benchmark dose limit, not a NOAEL. So the variability of the rodent population is already incorporated. To divide that by 10 means that our starting point is humans are ten times more sensitive, not less sensitive.

Keating said he used two uncertainty factors, one for inter-species and one for intra-species. The intra-species is for variability among humans.

Stelljes said the question was perhaps philosophical for all the times HEAC bases standards recommendations based on full data sets that incorporate variability in the test population. How much additional variability do we anticipate in the human population? Is it tenfold greater? You really need to ask that question every time. It shouldn't be a precautionary factor unless based upon some rational reason. If we don't think it's rational in this case, we should talk about it.

Stelljes said that is because of situations where we relied upon one data point, relying on a NOAEL or LOAEL. Maybe we have fifty animals so we have uncertainty around that level. You are taking the lowest dose and dividing by an uncertainty factor. In this case you are taking the variability of the test population and you are calculating a lower 5th percent slope on that. So you are already accounting for 95% of the variability of the animal test population. And then we are dividing that by another order of magnitude to protect humans. I don't think that is a reasonable default *pro forma* approach when we are looking at BMD-based data. It's a different paradigm; we need to evaluate it differently.

Keating said for those who may not have followed, the BMD approach was used on the rat bioassay data. That is where you take the observed responses, BMD model it to a certain effect level (5-10%) and then determine the 95% likelihood using the confidence intervals of the modeled data.

Steve Smith said this would effectively adjust the recommendation from a 1-ppm recommendation to a 10 ppm recommendation for a PEL. Stelljes said yes, that would be the impact—maintaining the STEL.

Cooper said an implication of Stelljes comments is that humans are more sensitive intra-species, which it is not in this case.

Stelljes said yes, since this is one of those situations where humans are less sensitive, why should we regulate a substance as though we were more sensitive.

Forest said what Stelljes was saying makes sense at first. But if you are right, I would think this would have been addressed in discussion of benchmark dosing in the last 30, 40 years.

Stelljes said he had been to many toxicology conferences where he had talked about it. But it comes down to the precautionary principle, we've always done it this way, and you have to prove to me why we have to change it. As opposed to, well, it doesn't make sense.

Forest said when you are working with a NOAEL, there has been tremendous discussion of safety factors, and do they reflect anything real. And they do, actually. What you are saying makes sense to me. Some of what uncertainty factors do represent when talking about NOAELs might be incorporated, but I don't remember ever seeing a discussion about that. There has been so much discussion of the subject with NOAELs, if there is a reason it doesn't apply to BMDs, then I think I might have encountered that. I'm torn.

Stelljes said all he could say is that Michael Sauer and the mathematician who developed BMD, in talking to both of them, neither one of them expected uncertainty factors to be put onto their numbers. The application of this has a life completely by itself not related to the reason why the initiators of the BMD concept developed the method. So it's the regulated community that has decided to continue to use the UF of 10 because no one will say you've been too protective so I'll sue you. That's why it's still around and people don't talk about it. Except in cases I've brought up where we are looking at cleaning contaminated materials for something that's much lower than naturally occurring concentrations. Metals for example. It

doesn't make much sense to use extra uncertainty factors because then you end up with a level that's below background. There are many real world examples where the toxicology is not borne out by the reality. I'd like to see this committee take the next step on how we evaluate the data using state of the art information.

Forest said he didn't rule out that Stelljes was right, but I would not want this committee to act based on this reasoning when no one else has been raising this rationale.

Stelljes said it would be nice to bring these ideas to a larger group and vet it. Cooper suggested the Society of Toxicology (SOT) as a venue, and Stelljes agreed.

Keating said he couldn't recall what the factor of ten animal to human uncertainty factor was based upon.

Stelljes said it is the lack of knowing whether more or less sensitive. It's the precautionary principle because we are not sure of intra-species variability. What I'm saying is if the rodent interspecies variability is already captured, how much more variable are humans. I have tried to give a presentation on this three or four times at SOT, but I've always been rejected because it is not seen as new toxicological information. So they won't even let me bring it up.

Spielman said it goes back to criteria for selecting the animals in the first place. There are species that are more sensitive and susceptible. Previous versions of HEAC have had discussion on uncertainty factors. How have these evolved? Primary answer I've gotten is that's the way it's done.

Forest said for NOAEL safety factors, they can't be really, really concrete but when people have looked at inter- and intra-species variation, they find that the tenfold factor is reasonably appropriate.

Stelljes agreed that something like 96% of chemicals studied they find that tenfold factor.

Forest said, so it is not drawn out of the air. It has been shown to work; it has a basis in fact, now.

Owens asked what the significance of the data on material damage in the first paragraph of the health based recommendation.

Keating said one effect seen in the Haufroid study, I think 2 to 3% hematocrit, but I'm not up on what is considered clinical. Here, on page 7, according to the paper it is significant. The study reads: "consistent with hemolysis observed in animals, however both reductions were within normal range clinical values."

Owens repeated, but when does a person get clinical?

Stelljes asked what percent damage is actually toxic. I'm not sure of the significance if the cohort is still within the clinical normal range.

Forest said the point is to set the PEL to a level where the effects don't get into a clinical range. Anemia is a condition that doctors try to treat.

Keating said the cohort was exposed for five years at 0.75 ppm. This is the only worker cohort study with this effect I found in the literature.

Stelljes said the exposure level was probably an estimate based upon snapshots of exposure and assuming

consistent exposure over time.

Keating said the study did have exposure concentrations and a lot of urinary measures which they might have used as air exposure surrogates.

Michael Bates said the PEL, whatever it may end up being, will the PEL be a combined value for both substances? This needs to be clarified.

Smith said the summary document will distinguish between the 2BE and 2BEA, but the value recommended for both is the same numerically. We debated internally whether to have two summary documents, one for each substance. But we put them together because these substances are so similar, providing the same hazards and recommended exposure levels.

Cooper asked, on page 10, which substance is being reported with 3 users? It makes sense to me that after our conversation and the PEL is set to separate them out so that it is clear. [After some discussion it was decided that the reference was to the top of page 9, and in this version of the document reference to the two substances had been separated out.]

Keating said he can bring back several pieces of information without a lot of additional work. One would be the clinical significance of hematocrit blood levels. I can do more on the factor of ten with a quick check on any other studies of intra and inter species variability ranges in blood hematocrit.

Bates said where do you want to go, one or ten? If you can support it maybe you can justify Stelljes new approach.

Cooper said he doesn't want to lose the concept, so he would recommend set it at ten, and then go back and validate it.

Forest said stick with standard methodology at 1 ppm and bring back supporting rationale for changes.

Stelljes said I think that's probably the better way to go, even though it was my idea. So we make sure we don't depart from standard unless we are pretty darn sure we have a reason.

Keating said while the recommendation is not final, for now it is 1 ppm, unless I can come back with a good rationale. Cooper argued for starting at ten ppm, so as not to lose the concept. Keating said he will come back, either way.

Smith said it was 1, with an asterisk, moving forward.

Methyl-isobutyl ketone (MIBK)

Keating said this is the first HEAC discussion of MIBK. There are different studies with different health endpoints: one developmental and the other kidney cancer in rats. Some compounds have a very specific mechanism in the male rat kidney known as 2-alpha globulin that is not relevant to humans. MIBK is one of those compounds. It has evidence of the 2-alpha mechanism in males; the females don't get 2-alpha, but they get chronic peripheral nephropathy (CPN) from MIBK. EPA is in the middle of reviewing tert-butyl ethanol (TBA) which has the same kind of toxicology. So, I propose to table MIBK and wait on that discussion and interpretation to learn from that for a future MIBK discussion. In the MIBK document I

quote from EPA draft tert-butyl study where it is acknowledged that some endpoints are not relevant for hazard assessment but others could be. Alternatively, OEHHA has prepared a TBA cancer risk assessment using the male rat kidney data. For discussion today, using standard uncertainty factors for occupational exposures a PEL of 7.5 ppm using the developmental study and a 0.33 ppm using a standard NOAEL of the CPN data in female rat.

Smith asked the committee their preference: wait till later in the year or come out with a proposal on one chemical sooner.

Forest said it made sense to discuss both chemicals, because it is a controversial postulated mechanism for both chemicals. The two should be discussed together. But the mechanism has been discussed for 30 years, so I'm not sure we have to wait for completion of the EPA process.

Keating said we can work from the IRIS draft while the comments are being reviewed by the IRIS board. Though there is a benefit of having the IRIS expert panel weigh in on it.

Cooper said he didn't want to hold up the HEAC process, and Forest said proceed with what we've got.

Smith said we can bring in the IRIS information at the next meeting if it is ready. Even if that information comes in later, significant data can be incorporated in the rulemaking, which, as you know, is not a short process.

Cooper asked Keating if he would come up with a tert-butyl acetate draft proposal. Keating said yes. Cooper said that, when the process reached the point of presenting the disabilities caused by the two chemicals, the two be separated so the consideration of each could be independently evaluated. Two separate summaries. One CAS number, one PEL recommendation.

Owens asked about the figure on the bottom of page 9. Keating said that was discussed earlier, on page 3, olfactory perception suppression. That study shows on the x-axis that seven subjects exposed to concentrations of MIBK, their thresholds went up compared to controls. They are being tested with an odorant after being exposed to MIBK. Looking at the basis for a STEL, the graphs for 100 and 200 ppm were similar, this sets a STEL threshold for this effect. The current STEL is *75* ppm, and the draft proposes 25 ppm.

Owens said so you think there is a higher odor threshold, the STEL would be based upon a changing odor threshold? Keating said right. At twice the concentration, the same response was seen. The STEL is based on a different endpoint than the PEL. The second line in the graph shows the test subjects returning to the baseline odor threshold over time after MIBK exposure has ceased. We don't have to address suitability of this endpoint for a STEL today.

Aluminum

Keating said the aluminum discussion was finalized last meeting. The summary was brought back to show the final document and particularly the table on page 7.

Unmack said this would have economic impact on users of aluminum oxide for blasting. Literature suggests that larger particle sizes are addressed by the current PEL. Lowering the PEL by a factor of 10 will affect these users of aluminum oxide for abrasive blasting. Each aluminum form had its own universe that was working with it. Each aluminum form--powdered aluminum, for example—changes how the body reacts.

Harrison agreed there are many problems with aluminum; in the literature there are cases of asthma and interstitial lung disease caused by aluminum oxide particles of various sizes. What are we regulating here when we say aluminum.

Unmack said initial HEAC summary was of aluminum and its soluble compounds. We were thinking of a handful of aluminum silicates and a lot of different aluminum oxides.

Smith said first HEAC draft had some forms at 2 and some at 1 but the group decided aluminum at last meeting that all forms should be at 1. We talked about solubles and insoluble forms

Unmack said one would not be anywhere near protective enough for erionite, another form of aluminum.

Forest said erionite should be regulated on its own, and have its own standard. Unmack agreed. Forest said it is like asbestos, fibrous, but about 800 times more toxic.

Cooper said we did not intend to cover all forms of aluminum - we don't want to address fibrous aluminum silicates such as asbestos and several other minerals. Is the PEL for aluminum oxides in various forms and sizes or anything with aluminum in it.

Unmack said it literally means aluminum and aluminum insoluble compounds, which I think is overly broad.

Cooper did not remember discussion about any soluble or insoluble compounds with aluminum in them.

Smith said the PELs and TLVs for metals are written this way. First the metals, and then the PELS for the metals' compounds. Sometimes they are limited to the soluble or insoluble forms. As we heard it, the group thought that all the forms should have the same PEL.

Stelljes noted some PELS differentiated between metal forms based upon toxicities.

Smith said previous draft had multiple values but committee recommended that forms should be all one value. I was hoping not to revise today. Consideration of the aluminum PEL has been going on for five years.

Cooper said so we are not having a discussion of feasibility at this point?

Smith said feasibility has been solicited on website for 5 years. We have indications from industry that

they may have concerns. They can bring them to us; they are not foreclosed from doing that.

Willhite disagreed with lumping all aluminum compounds together. For example, aluminum phosphide degrades to phosgene. Be very specific if you are doing aluminum oxide, aluminum hydroxide, metallic aluminum.. I've selected 50 relevant references from the 1172. I've highlighted the TLV verbiage in my submittal. I recommend reading the summary, introduction and conclusion at minimum. The reason I can't say if 1 mg/m^3 is a good number is those documents report subtle neurotoxicity cited in your documents. Many others dispute neurotoxicity; other organ systems affected, such as bone. Some studies show increased risk of ischemic heart disease—so there may need to be a dust number separate from the conventional aluminum oxide number. Once you figure out which organ system effect has the lowest LOAEL, then you can design a number. You can't lump them all together. What is needed is a rigorous systematic review of all of that literature.

Cooper noted HEAC had received the 250 page submittal yesterday.

Smith said multiple meetings had discussions setting different PELS for differing aluminum forms, but the last meeting had decided, at the very end of the last meeting, that it was better to have the same PEL for all.

Forest said there were things like erionite and aluminum phosphide that we can identify as chemicals that should be treated separately and otherwise regulated. We can distinguish other aluminum compounds that we are setting this PEL for. It's not particularly difficult to do.

Smith said this is not new language. The term "insoluble forms" is used not only for aluminum, but also for many metals. . We are just considering a change from the existing PEL of 10 and 5 to go to 1 total. Recommendations even from the previous HEAC committee, which at that point were at 2, if I recall, and we just went to 1 at the last meeting. Is the economic impact much different from 2 to 1?

Unmack said the aluminum oxide compound used in abrasive blasting might be impacted, as currently they are living with 10. Did not know what economic impact of 1 ppm will have on them.

Smith asked if a lot of that abrasive blasting exposure was respirable, so wouldn't that be a reduction of 5 to 1? Smith clarified that the recommendation from the last meeting was 1 total, to simplify the standard. The recommendation was all forms be at 1. I'm sure we will have more submittals on economics, but from HEAC, I'm trying to summarize what we heard last time.

Owens said he would rather separate out forms to be able to distinguish different health effects from different compounds. Lumping all together sets you up for questionable citations and questionable values.

Harrison disagreed: adopt the value of 1 ppm as proposed. He agreed with Smith that the current nomenclature for PEL listing of aluminum soluble forms is widely understood, measured and regulated for decades. I agree with using the physiological endpoint chosen. There are also other endpoints we could have looked at and at later point we could look at these other sensitive biological endpoints. I think we should

go ahead because almost all forms of aluminum are biologically active, and the basis for moving to 1 is the neurological endpoint among welders.

Stelljes said, looking at Table AC-1, I see seven different kinds of aluminum compounds. All separately regulated with individual PELs (2 to 10). I don't know why we think we could put it all together. None of these say what Smith said the definition was: "aluminum and insoluble" whatever. That is not listed for any of these 5155 chemicals.

Smith said we were trying to summarize what was said at the last meeting. I heard the recommendation to go to 1 for all forms. We are changing terms and eliminating some. We could use the old terms, including "aluminum welding fume," if that helps with clarity. But what we heard was to simplify the list of aluminum forms listed in 5155, and that they should all be at 1.

Cooper said he was at the last meeting, but was not recalling that conclusion. I thought, based on the welding fume discussions, that it had a different particulate size and was the one form we were looking at. I didn't understand that that applied to all of these, including the stearates, etc. If I had heard that I would have said something.

Spielman said, so the recommendation to go to 1 would be on the basis of elemental aluminum? Thereby we are setting a different standard for different molecular compounds.

Smith said if you look at what the ACGIH did, they used to have a list of 8 or 10 different breakouts of aluminum compounds. They consolidated to one TLV.

Spielman said if you go to a 1 across the board compared to a 5 respirable, you are probably significantly reducing exposures. Including for respirable.

Cooper said we want to base it on the toxicology. If particle size is reflective of toxicology, we should figure out a way to justify that within the table.

Willhite said, to clarify the TLVs, aluminum chloride and aluminum sulfate and others that were in the old TLV documentation were derived based not on data, but by analogy to their hydrolysis products: hydrochloric acid and sulfuric acid. There was no data to back that up; it was a judgment call by the TLV committee. ATSDR 2008 declined to set a value for aluminum inhalation minimal risk level because of uncertainties in the references you cite. So it is important to look at those other sources. Then you can make a judgment about where you are going. These substances have their own individual toxicity and bioavailability. You can't just lump them all together.

Unmack said he agreed with Smith's idea of having a floor that would be protective for a vast number of aluminum compounds. But we need to go further, because this change is going to have serious economic

impact in some sectors of our economy, such as abrasive blasting. This is something that you want to consider before you go forward.

Cooper asked if the list would come back to HEAC for a feasibility discussion or does that discussion not happen in the committee?

Smith said we've had five years of meetings that included some discussion of feasibility. We will continue to have more feasibility input if you like, but the intent is not to burden HEAC with that discussion. The Division ultimately has to make the call on feasibility, as we have done in the past. We've gotten input from HEAC, letters of submittal from stakeholders and made changes. Even after we initiate rulemaking, the Standards Board has made changes based on feasibility. There are other layers here.

Smith said, as spelled out in the HEAC process document, the committee's goal is primarily to look at the health effects. But this is also a forum to get stakeholder input on feasibility and to consider that in the whole process. We tried to get feasibility input in the previous HEAC/FAC process, but we've changed to the present format.

Leacox said there is an expectation that HEAC includes feasibility discussion. Is this just a forum for stakeholders to give you data? That is different from a forum in which that data is going to be discussed?

Smith said it will be discussed, but that is not going to hold up this process simply because there is a potential that some further feasibility data is going to come to us. At some point we need to move it along and that is what we are trying to do. We were trying to wrap up from the last meeting, but we can entertain more clarity on what we thought we heard.

Harrison said we have a two-part process. HEAC, advise Cal/OSHA on the PELs based upon a health-based assessment of available data. Then we should consider feasibility. As a committee member I separate the two. I'd welcome feasibility information. It has to be pretty concrete and specific information. We can consider that as we move ahead from here. Unless there is something new, I don't think there is anything I can say except I recommend a PEL of 1 unless there is some information that comes in that shows it's not feasible.

Cooper said he didn't have problems with that but the health effect question for me is for all these materials at 1, are we talking about the same toxicity. Or do we have some particle size issues, i.e., welding, where it justifies the 1 based upon the work, but not for the others. That's what I'm missing from the conclusion of let's make them all 1. I missed the part where we said the toxicology for all these matches the discussion we had for welding.

Harrison said that if we looked at sensitization as an endpoint, we would be at lower than 1. Based upon aluminum pot room workers. If we were to regulate on the basis of sensitization, we could have a discussion at a much lower level.

Unmack acknowledged that when he did the original research, he did not address some of the other health effects, as Harrison had stated.

Harrison said the pot room sensitization effect was different from welding and was from the aluminum exposure. [The opinion of several HEAC members was there was no aluminum pot room work currently in California.]

Cooper said he would argue to keep the list the way it was, and go with welding at 1.

Smith said welding fume is currently at 5. Are you proposing to lower welding fume to 1 and keep alkyls at 2? Alkyls are currently at a lower PEL than welding fumes, and you want to keep it at a higher PEL than welding fume?

Cooper said if there is a way to justify it, I'm all for it. I didn't get that, other than the change was making it simpler.

Smith said it wasn't a matter of making it simpler. What we heard was the committee thought all four should be at 1. Clearly there is no confusion on welding fume, so we will leave that at 1. I'm hesitant to put things back like stearates at 10 and alkyls at 2 because I heard at the last meeting that the committee wanted everything to go to 1.

Cooper asked if we had data to lower the alkyls and stearates.

Forest said it is not so much that. But there is data on aluminum very broadly. But data on any one particular aluminum compound such as stearates might not be available. There is no good reason to treat these compounds differently from other aluminum forms. But unless we have a demonstration that it is different, why would we treat it differently. I think that was the logic leading to the proposal for 1 for all forms.

Unmack noted that lead stearate is treated differently than other organo-leads because it is used as a soap and lubricant in industrial processes. Aluminum stearates would be soap-like. Toxicology of soaps has to be a whole lot different than alkyl metal toxicology.

Keating said ACGIH said the alkyls were particularly reactive—not the stearates. The alkyl groups.

Owens said organo-metals are usually more toxic.

Smith said we will go through what has just been submitted, but at this point he wanted to keep the last recommendation. We will put in welding fume for clarity, at 1. Let's see what Keating finds when he reviews the new material provided. When we do the formal rulemaking, we will certainly justify each of these changes as to why we moved forward with this number. We will make sure that this is supportable, as Forest mentions, utilizing the precautionary principle.

Cooper said we have several recommendations on the table. Change welding to 1. Leave the others alone

until we have data.

Smith said we were summarizing the recommendation from the last meeting, not soliciting new recommendations. We want to move forward with what we thought we heard from you. If we come up with information that we shouldn't move forward on some of these substances, we'll not do it. We think we have the data, but we'll revisit it.

Stelljes said assuming the other aluminum forms are less toxic, than the 1 is going to be health protective. It may be more burdensome than necessary from a feasibility standpoint. Using the precautionary principle presumes the other forms are just as toxic as the forms we do have data for. Right now that's what the proposal is.

Forest said, given that aluminum is really the issue, to the extent it is organic aluminum it is probably going to be more toxic. Except for aluminum phosphide which is a special case. Or erionate.

Cooper said given what we've just heard we should lower the PELs for all metals to 1 mg/m^3 because it would be more protective. But that is not what we are supposed to be doing. We are supposed to be looking at the individual players and coming up with the data that exists.

Stelljes asked, all under one CAS number? Answer: no. Stelljes said we just talked about each CAS number having its own value. Now we are saying we are only doing that sometimes, not all the time.

Cooper and Keating clarified that some of the aluminum listings like distearates had their own CAS number. Keating said he didn't know if every possible aluminum form had its own CAS number.

Stelljes said that if aluminum welding fumes and insoluble forms are one CAS # then I don't think there is an argument because it is all covered under the same CAS number—the products are the same. Different CAS numbers should be evaluated for their own toxicity, not used as an analogy to a different chemical with a different CAS number.

Cooper and Owens asked if welding fumes and pyro powders had CAS numbers.

Smith said we will take this under advisement. We'll make sure the summary data has substantial support for the recommendations. HEAC members can certainly look at what was recently submitted as well, and contact Keating with any comments. To a Cooper query about erionite, Smith noted asbestiform aluminum forms are not covered in the PELs under discussion right now.

Willhite asked for the conclusions and actions to be restated. Smith said we would continue with the summary based on the last meeting, adding for clarity aluminum welding fume, or that is, put it back in and set it at 1. We are going to double check our data and make sure that all these forms are supportable by the data that we have that these forms should also be at 1. If not we will come back to this committee and revisit

as something that shouldn't be at 1. So, it's back in Keating's basket, but committee members and stakeholders can still submit clarifying information for us to look at in more detail regarding the various forms. And stakeholders can still submit clarifying information for us to look at in more detail regarding the various forms.

PEL Prioritization

Keating pointed out the revised chemical priority list handout, but the meeting had run over time, so there was not much discussion. Keating said Cooper and he had devised this flow chart to priority rank chemicals so the P1 list could be repopulated. A PEL reduction of ten times was an important aspect of moving a chemical to P1.

Stelljes said chemical use data in California should be important in priority ranking chemicals.

Keating said he was attempting to access CERS data to help with that California use assessment.

Owens said the term "adverse endpoint" rather than "health endpoint" used in the flow chart might be problematic. Keating said he would look into that.

Adjourned: 3:47 PM