Received by the SAB Staff Office on October 23, 2012

Comments to full SAB Panel on "Draft Toxicological Review of Libby Amphibole Asbestos" (EPA/635/r/002a); Follow up to Chartered SAB's Written Comments and September 25, 2012 Teleconference

Elizabeth L. Anderson, Ph.D., ATS Fellow.

Exponent, Inc.

October 2012

This comment concerns two of the issues that require revision to the Panel's draft report to satisfy the directives of the chartered SAB regarding EPA's "Draft Toxicological Review of Libby Amphibole Asbestos. Among other topics, the chartered SAB asked for revisions of the Panel draft report concerning: 1) the controversial subject of whether reduced pulmonary function has been demonstrated to be casually related to localized pleural thickening ("LPT"); and 2) the absence in the draft report of an explanation as to Dr. Scott Ferson's non-concurrence. Attached are several emails concerning these two topics. These emails contain explanations provided by SAB Panelists during their deliberations. These SAB Panelist explanations are not posted on the SAB website, but were obtained on October 16, 2012 through a FOIA request. The emails provide additional information regarding the Panel's deliberation on LPT as a critical noncancer endpoint and reveal confusion regarding application of EPA guidance to selection of that critical endpoint. They also explain Dr. Ferson's non-concurrence.

In addition to providing the Panel's emails, this comment supplements my prior comments and explains that the Panel's conclusion regarding LPT does not reflect the weight of evidence and fails to provide EPA the requested advice on LPT. In my joint comments with Dr. David Hoel to the SAB Panel on April 9, 2012, we explained that the weight of evidence does not support a scientific conclusion that LPT is an appropriate critical endpoint because it is still *highly* debated in the medical/scientific community whether or not LPT impairs lung function. EPA would be taking a novel approach if it derives an RfC from an endpoint that has not been demonstrated to cause impairment, and this is especially true given the unprecedented step by EPA to formulate an RfC for an asbestos fiber.

1. Correspondence Amongst the Panelists Demonstrates Confusion; How to Apply EPA Guidance for Identifying Critical Endpoints to Localized Pleural Thickening.

I have been trying to understand why the pulmonologists on the Panel are supporting statements in the draft report (8/30/12 version) that are at odds with the clear weight of scientific literature. Recent panelist emails seem to provide two explanations in response to public comments on this issue.

- First, the panel may have discounted the accepted clinical understanding that LPT has not been shown to impair lung function and instead may be trying to apply an undefined "public health" meaning to the term "adverse effect."
- Second, one email clarifies that the "association" between LPT and changes in lung function that the Panel's draft report discusses is an "independent"

> association because studies have not proven a direct causal association. In other words, in the Panel's view, even if the lung function changes are due to undetected early asbestosis or obesity, the concurrent presence of LPT provides a sufficient independent "association" to support use as an endpoint under at least one panelist's explanation. However, this is not how IRIS determinations of the critical endpoints are made.

On the first point, by discounting the accepted clinical understanding that LPT has not been shown to impair lung function, the Panel has failed to provide EPA with the requested guidance on LPT as the critical effect. We recommend that the Panel provide EPA with an objective and clinically and scientifically based response that does not gloss over unresolved scientific issues. For example, please see the attached email explanation dated July 28, 2012, suggesting a sharp distinction between application of clinical experience and the standard of review that the Panel is applying to the risk assessment. This is an excerpt of that email:

> *"It may be helpful for the EPA to more fully explain Rfc version of health effect vs clinical disease. ATS document focused on clinical asbestos-related disease. Clinicians / others are so used to reassuring patients that plaques are no big deal, don't affect lung function (esp as typically past exposure can't do anything about), that they may need an extra reminder as far as Rfc / the public health perspective. It took me a while to remember this after 'minimizing' plaques with individual patients for so long."*[1]

This email seems to suggest that clinical experience and the clear findings of the American Thoracic Society (ATS) are not being applied by the Panel, including the ATS finding for patients with pleural plaques that "decrements when they occur are probably related to early, subclinical fibrosis" and "even so, most people with pleural plaques alone have well preserved lung function." It is incorrect for clinical findings to be disregarded in an evaluation of "adverse effects." Instead, under controlling EPA guidance, an "adverse effects" evaluation should be based upon clinical observations and the very type of epidemiological literature upon which the ATS based its conclusions.

EPA defines adverse effect as *"[a] biochemical change, functional impairment, or pathologic lesion that affects the* ***performance*** *of the whole organism or reduces an organism's ability to respond to an additional environmental challenge*."[2] An adverse effect must have biological significance such that it:

> "***is likely to impair the performance or reduce the ability of an individual to function or to respond to additional challenge***

[1] Email sent by Dr. Redlich on July 28, 2012. (Attachment 1).
[2] Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Part F, Supplemental Guidance for Inhalation Risk Assessment (Jan. 2009) at 9, emphasis added, *available at* www.epa.gov/oswer/riskassessment/ragsf/pdf/partf_200901_final.pdf.

> *from the agent. Biological significance is also attributed to effects that are consistent with steps in a known mode of action. Statistical significance quantifies the likelihood that the observed effect is* ***not due to chance alone. Precedence is given to biological significance, and a statistically significant change that lacks biological significance is not considered an adverse response****."*[3]

Under the above EPA guidance and definitions, adverse effect is determined based upon clinical experience and epidemiological data, and depends upon a finding of a clinically significant impairment. As explained in my April 9, 2012 comments and in other public comments, no finding of clinically significant impairment due to LPT can be supported based upon the weight of the scientific literature.

Regarding the second point, the EPA guidance makes clear that a coincident or independent association among effects is not enough to establish an adverse effect. Thus, any supposed "independent" association is insufficient to support a conclusion that LPT causes lung impairment. Instead, a finding of an "adverse effect" requires more than chance or an independent association among separate effects; we are looking for an impairment that is causally related to the pleural plaques in order for LPT to be a critical endpoint. One has not been demonstrated. Nevertheless, the following email dated June 27, 2012 suggests the opposite: that a biomarker alone or an independent relationship that only suggests an "association" is sufficient to support the endpoint. I find this interpretation to be at odds with the general application of EPA guidance for establishing a critical endpoint.

> *"With regard to lung function changes, the point of my remarks is that* ***regardless of whether or not LPT is associated with observable lung function changes****, it is in and of itself an irreversible pathological change in tissue structure. Risk assessment guidelines identify that endpoint as a suitable (and indeed, fairly severe) endpoint for use in risk assessment, regardless of whether functional changes are observed as a result of or associated with that finding. The panel subsequently discussed the question of whether, in addition to LPT, the amphibole exposures were also associated with observable lung function changes in the dose range of interest, and it was concluded that they were. It appears that LPT findings are not invariably associated with observable lung function changes, or vice versa: how much of this is due to relative insensitivity and imprecision of these clinical evaluations, or merely to the fact that*

[3] EPA. A Review of the Reference Dose and Reference Concentration Process (Dec. 2002) at 4-11, emphasis added,
available at http://www.epa.gov/raf/publications/review-reference-dose.htm ("a statistically significant change that lacks biological significance is not considered an adverse response.").

> *they are seldom done simultaneously on the same subject, is unclear. However, the risk assessment conclusions are simpler:* ***both LPT and lung function changes are separately demonstrable effects of exposure to amphiboles, which may be considered independently in determining dose response relationships for adverse effects***."[4]

Again, the full email is attached so it can be reviewed and assessed in full. The panel member author of this email appears to be saying that regardless of whether LPT is associated with observable lung function changes or not (and he seems to concede that this is an unresolved question), it is sufficient, for purposes of establishing LPT as an endpoint, that LPT and lung function changes are "separately demonstrable" effects of exposure to amphiboles. As noted in the portion of the EPA guidance cited above, this showing is insufficient to establish LPT as an endpoint.

It seems that these emails acknowledge precisely the point that Dr. David Hoel made to the chartered SAB on September 25, 2012: that scientific literature has <u>not</u> established a causal relationship between pleural plaque and any lung function change. Furthermore, the attached email confirms that pulmonologists offer clinical advice consistent with this scientific understanding. The experts seem to be in accord on this issue. The confusion lies in the Panel's erroneous application of the EPA policy regarding what is an "adverse effect" either by applying a novel undefined "public health" standard that disregards clinical and scientific knowledge or by inflating the significance of a biomarker. EPA has asked for the SAB's input on the scientific point: "Please comment on whether the selection of this critical effect [LPT] is **scientifically supported** and clearly described."

EPA has not asked for SAB guidance on EPA's existing policy for critical effects nor has EPA asked the SAB to expand EPA's policy on adverse effects. EPA would be best served by an objective and clinically and scientifically based response that does not gloss over unresolved scientific issues.

2. Dr. Ferson Explained in an Email Precisely Why He Refused to Concur with the Report.

The chartered SAB asked questions about the basis of Dr. Ferson's non-concurrence with the Panel's draft report. Emails from Dr. Ferson were included in the FOIA response identified above. (Attachments 3 and 4). These indicate his concern about EPA discarding large amounts of data and its acceptance of "much more arbitrary and less reliable" results because of the limited data employed. Though raised in the context of the Libby data, Dr. Ferson's concern and rationale seem equally applicable to the Rohs data, where EPA similarly discarded large amounts of data. Thus, Dr. Ferson's concern has implications for both the IUR and the RfC. As the chartered SAB made clear, the Panel's draft report should be revised to include a clear and accurate statement of Dr. Ferson's non-concurrence and the reasons for it. Alternatively, the

[4] Email from Dr. Salmon, June 27, 2012 (emphasis added). (Attachment 2).

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Report should be revised to advise that EPA make use of the larger amount of available data to improve the validity of the analysis.

Thank you for your time and attention to these short supplemental but very important comments.

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ATTACHMENT 1

Received by the SAB Staff Office on October 23, 2012

From: Redlich, Carrie
To: Diana-M Wong/DC/USEPA/US@EPA; Agnes Kane
Subject: Word of explanation re LPT associated with increased risk meso, lung ca
Date: 07/28/2012 09:04 PM
Attachments: asb pleural meso[3].pdf
asb plaques lung cancer.pdf
Reid Addit risk meso wittenoom OEM 2005.pdf

Agnes/ Diana
I found this in my outbox – not sure if sent earlier in the week- may be duplicate email
carrie

Agnes / Diana
I thought I should add a word of explanation for deleting a sentence that generated so much attention (below - I didn't write it) and my other more minor edits.
While the ATS asbestos document does say LPT associated with increased risk asbestosis, ca, meso, it cites only 2 references to support LPT associated with increased risk of mesoth and lung cancer (beyond exposure history). Most clear, and what we discussed at our meeting and prior calls, was that LPT associated with reduced lung function, which a number of well done studies document. We suggested EPA further highlight this literature and added a few additional references. Not a big deal / change.
I had been uncomfortable with LPT being predictive / associated with increased risk of meso, lung cancer, so I had done some searches of the epi literature (see attached). The question is complicated by 1) confusion if referring to plaques as a marker of asbestos exposure vs increased risk beyond estimated exposure (the real Q), and 2) studies have mostly used occupational history for exposure assessment.
One of the better articles (Reid) and brief lit search attached. (Reid already cited by EPA somewhere. Don't think EPA needs to add any refs).
Bottom line – while ATS statement likely correct, **there's not much evidence to support LPT and increased risk meso, lung ca (beyond exposure),** and as mentioned, no need to go there. It's confusing and nonmalignant changes sufficient justification as endpoint, and it's just opening up EPA for criticism. This is referring to LPT and risk of meso, lung cancer. There is good data that supports LPT and reduced lung function. (my edits tried to clarify this).
Sorry didn't bring this up on the call – I was hesitant to start a whole discussion about. I looked over articles etc more carefully when doing edits and realized that while "associated" better than "predictive", even better to omit.

As you know, asbestos differs somewhat from pollutants such as ozone, as there are well known clinical entities caused by asbestos. It may be helpful for the EPA to more fully explain Rfc version of health effect vs clinical disease. ATS document focused on clinical asbestos-related disease. Clinicians / others are so used to reassuring patients that plaques are no big deal, don't affect lung function (esp as typically past exposure can't do anything about), that they may need an extra reminder as far as Rfc / the public health perspective.

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It took me a while to remember this after “minimizing” plaques with individual patients for so long.
Hope this helps.
Carrie

On 7/25/12 6:52 PM, "Carrie Redlich" <carrie.redlich@yale.edu> wrote:

“Additionally, the presence of LPT itself is predictive of risk for other asbestos-related diseases, including asbestosis, mesothelioma and lung cancer, a point that the EPA should include.”

Carrie A. Redlich, MD, MPH
Program Director, Yale Occupational and Environmental Medicine
Professor of Medicine
Occupational and Environmental Medicine and
Pulmonary and Critical Care Medicine
Yale School of Medicine

YOEMP
135 College St, 3rd floor
New Haven, CT 06510
Tel: 203-737-2817 Fax 203-785-7391
Cell Phone: (b) (6) [redacted]
carrie.redlich@yale.edu

The information contained in this message may be privileged and confidential. If you are NOT the intended recipient, please notify the sender immediately with a copy to hipaa.security@yale.edu and destroy this message. Please be aware that email communication can be intercepted in transmission or misdirected. Your use of email to communicate protected health information to us indicates that you acknowledge and accept the possible risks associated with such communication. If you do not wish to have your information sent by email, please contact the sender immediately.

Received by the SAB Staff Office on October 23, 2012

ATTACHMENT 2

Received by the SAB Staff Office on October 23, 2012

From: Salmon, Andy@OEHHA
To: Diana-M Wong/DC/USEPA/US@EPA
Subject: RE: Public Comments Posted on Our Website
Date: 06/27/2012 05:13 PM

Having taken a look at these comments, I do need to respond to their mischaracterization of my earlier remarks about LPT as a toxicity endpoint. They appear to think that I was discounting the possibility that LPT was associated with changes in lung function. I never said anything of the sort. In the first place, the discussion about where LPT stands on the overall mechanistic pathway started in the context of mesothelioma rather than lung function changes. The general conclusion of the panel (with which I agree) is that there certainly are common elements to the causative pathways for mesothelioma and LPT, but it is not correct to see LPT as an obligatory precursor to mesothelioma, i.e. not all LPT lesions will progress to mesotheliomas and not all mesotheliomas arise by progression of LPT lesions. But both types of lesion arise as the result of the cellular damage induced by the persistent fibers and other associated effects. With regard to lung function changes, the point of my remarks is that regardless of whether or not LPT is associated with observable lung function changes, it is in and of itself an irreversible pathological change in tissue structure. Risk assessment guidelines identify that endpoint as a suitable (and indeed, fairly severe) endpoint for use in risk assessment, regardless of whether functional changes are observed as a result of or associated with that finding. The panel subsequently discussed the question of whether, in addition to LPT, the amphibole exposures were also associated with observable lung function changes in the dose range of interest, and it was concluded that they were. It appears that LPT findings are not invariably associated with observable lung function changes, or vice versa: how much of this is due to relative insensitivity and imprecision of these clinical evaluations, or merely to the fact that they are seldom done simultaneously on the same subject, is unclear. However, the risk assessment conclusions are simpler: both LPT and lung function changes are separately demonstrable effects of exposure to amphiboles, which may be considered independently in determining dose response relationships for adverse effects.

From: Diana-M Wong [mailto:Wong.Diana-M@epamail.epa.gov]
Sent: Monday, June 25, 2012 11:32 AM
To: Diana-M Wong
Subject: Public Comments Posted on Our Website

Dear Panel Members,

A set of public comments submitted by Karl Bourdeau of Beveridge & Diamonds is posted on our website for your consideration. The link is provided below:

http://yosemite.epa.gov/sab/sabproduct.nsf/MeetingCal/DE16F40DF2BE9271852579FB0054C2BF?OpenDocument

The pdf file is also attached.

(See attached file: Bourdeau June 25 no sig.pdf)

Sincerely,

Received by the SAB Staff Office on October 23, 2012

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

Received by the SAB Staff Office on October 23, 2012

ATTACHMENT 3

Received by the SAB Staff Office on October 23, 2012

From: (b) (6) [redacted]
To: Katherine Walker; Pennell, Michael; Salmon, Andy@OEHHA (Andy.Salmon@oehha.ca.gov); Julian.Peto@lshtm.ac.uk; Diana-M Wong/DC/USEPA/US@EPA
Subject: Re: Reminder: Response to Question 3, Section 3.2.6.3
Date: 08/03/2012 02:20 PM
Attachments: asbestos_july.doc

Katy:

What you're thinking of was in the email the morning of the discussion. I've attached the latest version of that, but it doesn't reflect Andy's comments.

Apparently, everyone is happy if we just "acknowledge" that something we recommend is "difficult". I think that gives them an out to not do it. As I mentioned, I'm not really inclined to do this too obviously, because it really just says, "okay, okay, don't do this if you don't want to." But what kind of a review is that? If they didn't want to know what we actually recommend, why'd they bother to ask?

About bounding the dependence, I think they actually should do it, or, as you said, at least marshall some kind of evidence for their assumption. That they did neither is an obvious deficiency that they seem disinclined to correct.

Regarding the other matter, about re-including the early data they ignored, you may thing that was a reasonable decision, but I really don't think so. It almost certainly colored their results to be much more arbitrary and less reliable than they should be. That they made no serious effort to save those data is laziness at best. Maybe they think it's "difficult" to do it right, but--let's be honest--we can't really tell whether this is true because they didn't try.

We've now given them a few options to explore on both issues. Should we really say now they don't need to do anything?

Scott

On Thu, Aug 2, 2012 at 2:14 PM, Katherine Walker <KWalker@healtheffects.org> wrote:

> She wants us to soften the language in the draft about the interval analyses. I need to look at it again –but it would help if you could look also and see if there is anything more specific in the advice we give them about what to do.
>
> I thought you had acknowledged the difficulty but maybe that was in your email comments and we need to incorporate them.

At some point, I need to discuss with you the comments I kept hearing "well, this is just for the risk analysis. (Medical practice is another matter)" Like this is somehow not real life. I bit my tongue, but I'm a bit disturbed.

What are we in, a parallel universe?

katy

From: (b) (6) [redacted]
Sent: Thursday, August 02, 2012 1:46 PM
To: Katherine Walker
Subject: Re: Reminder: Response to Question 3, Section 3.2.6.3

Katy:

I presume you're taking the lead on this. Let me know if you want to talk, or if you have an assignment for me.

Scott

On Thu, Aug 2, 2012 at 11:17 AM, Diana-M Wong <Wong.Diana-M@epamail.epa.gov> wrote:

Dear All,

As a reminder, the revised text is due tomorrow. Thank you very much.

Diana

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

Received by the SAB Staff Office on October 23, 2012

----- Forwarded by Diana-M Wong/DC/USEPA/US on 08/02/2012 11:15 AM -----

From: Diana-M Wong/DC/USEPA/US
To: Katherine Walker <KWalker@healtheffects.org>, (b) (6) <sandp8@gmail.com>, scott@ramas.com, "Pennell, Michael" <mpennell@cph.osu.edu>
Date: 07/26/2012 03:09 PM
Subject: Response to Question 3, Section 3.2.6.3

Scott, Katy, and Mike,

Per Panel discussion on issue 7 of EPA's comments, some revision to the response to Question 3 on page 39, line 12-27 is needed.

Please revise the text and send me the revised text by Friday, August 3rd. Thanks.

Diana

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

Received by the SAB Staff Office on October 23, 2012

ATTACHMENT 4

Received by the SAB Staff Office on October 23, 2012

From: Diana-M Wong
To: (b) (6) scott@ramas.com
Cc: Kane, Agnes
Subject: Fw: SAB Libby Amphibole Asbestos Report
Date: 08/29/2012 12:58 PM

Scott,

I have not heard from you if you plan to send me suggested changes. I have left voice mail in your office.

We need to submit the report for review by the full SAB. Thanks.

Diana

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

----- Forwarded by Diana-M Wong/DC/USEPA/US on 08/29/2012 11:36 AM -----

From: Diana-M Wong/DC/USEPA/US
To: (b) (6) [redacted]
Cc: "Kane, Agnes" <agnes_kane@brown.edu>
Date: 08/27/2012 05:58 PM
Subject: SAB Libby Amphibole Asbestos Report

Scott,

Given that you are not available till Wednesday, I appreciate you provide any suggested changes by noon Wednesday, August 29. Otherwise, I need to indicate in the report that you do not concur.this report.

Diana

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

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▼ Diana-M Wong---08/27/2012 03:34:53 PM---Scott, The draft text you referred to reflects the full responses to Question 3 and Question 4 in Se

From: Diana-M Wong/DC/USEPA/US
To: (b) (6) [redacted] >
Cc: Katherine Walker <KWalker@healtheffects.org>, Katherine Walker <kdwalker1206@hotmail.com>, "Pennell, Michael" <mpennell@cph.osu.edu>, Julian.Peto@lshtm.ac.uk, "Salmon, Andy@OEHHA (Andy.Salmon@oehha.ca.gov)" <Andy.Salmon@oehha.ca.gov>, "John Neuberger" <JNEUBERG@kumc.edu>
Date: 08/27/2012 03:34 PM
Subject: Re: Reminder: Concurrence on SAB Libby Amphibole Asbestos Report

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Scott,

The draft text you referred to reflects the full responses to Question 3 and Question 4 in Section 3.2.4.3 on page 21 and 22, respectively.

Do you have any suggested changes?

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone:(202) 564-2049

▼ SandP8 ---08/27/2012 03:06:55 PM---Diana: It is the first day of classes today, and am finding it difficult to be

From: (b) (6) [redacted]
To: Diana-M Wong/DC/USEPA/US@EPA
Cc: Katherine Walker <KWalker@healtheffects.org>, Katherine Walker <kdwalker1206@hotmail.com>, "Pennell, Michael" <mpennell@cph.osu.edu>, Julian.Peto@lshtm.ac.uk, "Salmon, Andy@OEHHA (Andy.Salmon@oehha.ca.gov)" <Andy.Salmon@oehha.ca.gov>
Date: 08/27/2012 03:06 PM
Subject: Re: Reminder: Concurrence on SAB Libby Amphibole Asbestos Report

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Diana:

Received by the SAB Staff Office on October 23, 2012

It is the first day of classes today, and am finding it difficult to be thorough in my review of the document you sent. I cannot always observe the deadlines that you set and inform me about.

I do *not* concur with this statement in the letter:

The SAB supports the selection of the Libby worker cohort for the derivation of the inhalation unit risk (IUR) and agrees that the use of the subcohort post-1959 for quantification is reasonable due to the lack of exposure information for many of the workers in earlier years. The SAB finds it appropriate to use lung cancer and mesothelioma as endpoints for the derivation of the IUR. However, the SAB recommends a more detailed discussion and justification of how the use of mortality data rather than incidence data may have resulted in an undercount of cases of lung cancer and mesothelioma.

I thought I was paying close attention, but did not notice until now that earlier language had been so watered down to be a complete capitulation to what I continue to believe is a flawed idea.

I don't think I'm merely being grumpy here. Perhaps someone can talk me down, but I'm a bit surprised and disappointed. Unfortunately, I am very busy this week. I may be able to revisit this on Wednesday afternoon.

Regards,
Scott

On Mon, Aug 27, 2012 at 10:41 AM, Diana-M Wong <Wong.Diana-M@epamail.epa.gov> wrote:

Dear Panel Members,

As a reminder, your response regarding concurrence on the revised draft SAB report is due today. Please reply to this e-mail by **COB today (August 27)**.

Thank you very much.

Sincerely,

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone: (202) 564-2049

Received by the SAB Staff Office on October 23, 2012

----- Forwarded by Diana-M Wong/DC/USEPA/US on 08/27/2012 10:27 AM -----

From: Diana-M Wong/DC/USEPA/US
To: Diana-M Wong/DC/USEPA/US@EPA
Date: 08/22/2012 10:44 AM
Subject: Concurrence on SAB Libby Amphibole Asbestos Report

Dear Panel Members,

Attached please find the revised panel report on review of Libby Amphibole Asbestos assessment. This draft reflects comments submitted by Panel members and discussions on the conference call on July 25.

(See attached file: Asbestos Report 8-20-12.docx)

I hope that you will agree that the draft now captures the Panel's advice to the Agency in clear, and consistent language. At this time, Dr. Kane and I are seeking your final concurrence on the revised draft so that the report can be forwarded to the full Science Advisory Board for discussion and (hopefully!) approval on a September 25 public teleconference.

Please reply to this email **no later than the morning of Monday, August 27,** and indicate that you either:

1) Concur;
2) Concur with minor editorial corrections; or
3) Nonconcur.

Thank you for all your hard work.

Sincerely,

Diana Wong, Ph. D., DABT
Toxicologist and Designated Federal Officer
USEPA
Science Advisory Board Staff Office
MC: 1400R
1200 Pennsylvania Ave, N.W.
Washington, DC 20460

Phone: (202) 564-2049