RE: RIN 1219-AB24: Proposed Rule – Asbestos Exposure Limit

November 15, 2005

The R. T. Vanderbilt Company, Inc. (Vanderbilt) supports the key provisions of the captioned rule and notes that the expressed scope of the rule does not include a change in the definition of asbestos. Further, the proposed rule relies upon the Occupational Safety and Health Administrations’ (OSHA) asbestos risk assessment which Vanderbilt presumes includes the 1992 OSHA review and ruling on amphibole cleavage fragments (57 FR 24310-24311). For this reason, Vanderbilt had not intended to provide comment beyond that submitted during the ANOPR phase of this rulemaking. Recent submissions into the rule making record, however, prompt additional comment.

Several submissions encourage the Mine Safety and Health Administration (MSHA) to broaden the definition of asbestos to include elongated amphibole cleavage fragments, if not during this rule making, during a subsequent MSHA rulemaking. These requests are linked to convenience (the use of simplistic fiber counting criteria to define asbestos and asbestos risk), to risk association (cleavage fragments are observed in asbestos exposures hence they may be equally implicated in the same disease end points) and on theoretical grounds (because of the same chemistry, biopersistent, and of respirable size, elongated amphibole cleavage fragments are assumed to pose the same risk as asbestos). Vanderbilt believes none of these theories are supported by scientific evidence—however well intended.

The recalculation of non-asbestos material “as asbestos” is no more supported today than it was in 1992 when OSHA addressed the regulation of nonasbestiform anthophyllite, tremolite and actinolite as asbestos. Thus, Vanderbilt cautions MSHA to ensure a thorough review of the science in this area if there is serious consideration to regulating non-asbestos as asbestos in this or any subsequent actions by the Administration. Most, if not all, of the authoritative literature on this subject has been submitted into the MSHA record and is also available through the cited OSHA rule.

Vanderbilt supports the regulation of any mineral particulate as stringently as asbestos if it is shown to pose the same risk. Vanderbilt does not, however, support mineral mischaracterization in the name of convenience or misinformation. Exposures should always be properly identified and called by their proper names. This is critical to accurate and meaningful risk assessment.
Since a change in the regulatory definition of asbestos would likely require an updated risk review, coordination with other government agencies and new rulemaking, our comments in this area are cautionary only. Given the impact of asbestos definitional changes, justification would certainly have to be demonstrated.

We also note that several comments submitted into the record allege an asbestos risk for Vanderbilt talc workers. Such allegations are not uncommon because our talc workers play a pivotal role in what is commonly referred to as “the amphibole cleavage fragment issue”. As a result of this focus, Vanderbilt talc is one of the most studied mineral products in the world from both a mineralogical and biologic perspective. Beyond numerous human health studies (both mortality and morbidity), Vanderbilt talc has also been tested in animal and cells studies against asbestos. These studies consistently show no carcinogenic response to Vanderbilt talc (in whole or part) while asbestos, tested under the same test conditions, consistently does.

Since the health experience of Vanderbilt talc workers has once again been raised (presumably to argue for asbestos definitional changes), we would like to call your attention to our prior ANOPR oral testimony. For convenience, we have appended the MSHA transcripts of that testimony. This testimony was provided during the MSHA public hearing held in Canton, New York on May 29, 2002. The object of this testimony was to relay updated health information regarding Vanderbilt talc miners and millers. Supporting documents (listed in the testimony) were provided at that time as well. Vanderbilt believes this testimony speaks directly to those who mistakenly believe exposure to Vanderbilt talc poses an asbestos-type risk (mineralogy issues aside).

Thank you for your attention to this matter.

Very truly yours,

R. T. VANDERBILT COMPANY, INC.

John W. Kelsc, Corp. Industrial Hygienist
Manager, Corporate Risk Management Dept.
TRANSCRIPT OF PROCEEDINGS

ASBESTOS LEVELS IN MINING FACILITIES

Pages: 1 through 60
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other than 3 to 1, and use populations --

MR. PETRIE: Excuse me one second. Can you speak up a little louder. We have some noise out in the hallway there. Some of the individuals are having difficulty hearing.

MR. KELSE: Okay.

MR. PETRIE: Thank you.

MR. KELSE: So at any rate, I will comment on those questions. But as you heard this morning, for decades, the ore from this mine has been accused of containing asbestos, and more importantly, as imposing an asbestos-like risk.

I really won't address the mineral issues, although I do have some slides. If you want, I can go through some of those, but I think it's pretty clear by now that the industrial grade talc that's mined at Vanderbilt isn't, in fact, an asbestos-containing material. I've left some supporting documents on that topic.

Because it's been suggested that the health experience of these talc miners reflects an asbestos-type risk, however, and because regulatory agencies have been periodically encouraged to regulate it has as asbestos, whether it contains asbestos or not, it's important to ask whether the health experience of these miners really is
reflective of an asbestos risk, mineralogy aside.

So to address that, I brought along some slides.

I'll pretty much stick to a prepared script so I don't stray. It's all too easy for me to do that. I can go off on tangents on this topic. I don't want to do that. I want to keep this to about 20 minutes and run through these slides.

First, what I'd like to go over, I'll go over the facility's pulmonary cancer experience. Remember, I'm talking about Vanderbilt talc here. I didn't work for Loomis. I didn't work for International. I don't know what their experience was. I'm talking about Vanderbilt. The only talc mining operation currently in this region. There are no others, just Vanderbilt. Then I'll briefly address the non-malignant respiratory disease experience.

(Slide presentation.)

MR. KELSE: This is a very busy table and very difficult to see. My other slides will be a lot easier to make out than this. But it's an extremely important slide because it reflects the most up-to-date breakdown of lung cancer deaths that we have among everybody that had ever worked at Vanderbilt talc.

MR. PETRIE: If you can excuse me for just one more second, let me see if we can turn these front lights off so we can better see them.
MR. KELSE: Sure. I don't know if I can focus that. I'll try.

(A short recess was taken at 9:57 a.m.)

MR. PETRIE: We'll go back on the record.

MR. KELSE: Are we back on?

MR. PETRIE: Yes.

MR. KELSE: Again, I apologize for this slide. It's extremely difficult to read, but it does reflect the most up-to-date breakdown of lung cancer deaths that we have. This covers anyone who had ever worked in the Vanderbilt mine or mill for any length of time since its opening in 1948 through 1989. That's a total of $18 over a 42 year period.

Now over the years, there has been no less than six mortality studies of this relatively small group of miners. So it pretty much places them among the most studied miners in the world. The 31 cases listed here does show an overall excess of lung cancer at approximately two and a half times the expected rate.

This a moderate, but significant excess, and one that is seen in all of the studies. If you look no closer than this, you might conclude that the exposure to this talc, whatever it contains, is likely responsible for these lung cancer deaths. Just as excessive exposure is linked to
lung cancer deaths, the belief that this talc poses an asbestos-like risk originated from these studies.

However, to truly establish a causal association, you need to look a little closer. And when you do, you'll see some very interesting things. One of the first things that jumps out at you is the much higher number of cases among miners versus millers. That's important because, as you've heard others mention here, dust exposure over the years show overall dust levels to be about the same in the mine and the mill with some historical reports showing higher dust in the mill.

There are slightly more millers than miners, about 15 percent more, who ever worked at this mine. And the average years worked for both groups is similar. So if the cancers are linked to the dust exposure, we would expect to see more cases among millers. But that's not what we see.

There is also a very high percentage of cases with very minimal dust exposure time or tenure on the job. In fact, 55 percent of all of the cases worked less than a year. Forty-five percent worked less than six months. And you'll see cases with one day, four days of exposure to the talc in their entire working lives.

If the dust is so potent as to cause lung cancer with such minimal exposure -- one day, four days, we would
certainly expect to see those exposed longer to show even higher lung cancer rates, but we don't. In studies of asbestos workers, we do.

Also, smoking histories -- always important whenever lung cancer is being studied, was obtained for a case control study. The case control study ran to 1985. For these lung cancer deaths, every case was a smoker. For deaths after 1985, we don't have reliable smoking histories, but I wouldn't be surprised if every one of those lung cancer cases were smokers as well.

Just as importantly, the researchers found that 73 percent of the non-cancer cases, the controls used in the study were also smokers. So in other words, we've got a lot of smokers in this mining population.

This table gives you an idea of how prevalent smoking has been among these miners compared with national norms. Our smoking records are less reliable prior to 1980, but I'm sure the rate was equally disproportionate -- about twice the national average.

Some researchers feel smoking alone could not account for all the excess. Others feel strongly that, indeed, it could. That it is, in fact, the more plausible explanation. But whether smoking, in whole or in part, is the reason for the persistent cancer excess, the next

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observation, I think, is very key.

The most recent mortality study included an analysis that all prior studies did not. That was an historical dust exposure assessment. This assessment showed that the cumulative dust exposure from the lung cancer deaths was 31 percent below the dust exposure for all decedents. In other words, we see an inverse dust exposure response relationship that further confirms what was suggested from the 10-year data, or time on the job experience.

In asbestos exposed workers, those with increased cumulative exposures do show increased lung cancer rates. In other words, you do see an exposure response relationship. You do not see that in Vanderbilt talc workers.

I believe this is about as strong as epidemiology gets short of a no-excess finding when it comes to cause/effect determinations.

(Slide presentation.)

MR. KELSE: This is an interesting slide. Also, one I need to apologize for. It's very hard to see this. What this does is compares lung cancer in non-malignant respiratory disease mortality among Vanderbilt talc workers, and Vermont talc workers.
Now I know it's difficult to compare one epidemiology study with another, but the comparison here, I believe, is pretty reasonable. Both groups have similar number of people; similar exposure years; similar overall dust levels; silica exposure isn't an issue in either study. And when you look only at the talc workers in both groups with more than one year exposure, the overall lung cancer rate is no different. In regard to non-malignant respiratory disease, it's actually lower in New York.

I put this comparison up because some of the mineral components in New York talc, incorrectly characterized as asbestos by some, or just bad as asbestos by others, aren't present in Vermont. So it doesn't appear these controversial mineral components make much difference. Incidentally, the moderate lung cancer excess in Vermont talc workers was not attributed to the dust by the researchers, which was NIOSH in this case. It turns out that there was also an inverse exposure response seen in Vermont. So factors other than the dust were cited as the likely cause of the lung cancers observed.

Well, beyond human mortality studies, it's always good to have animal study or two that supports or doesn't support the epidemiology. This table reflects the effects of a rat pleural implementation study by Moral Stanton of

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the National Cancer Institute. Dr. Stanton was testing the theory that morphology particle dimension was most key to fiber toxicity, if not the only consideration.

It turns out that among all the samples Stanton tested, I believe 72 in all, carefully measuring the particles in each samples, he tested an off-the-shelf sample of Vanderbilt talc as well as platy talc. As you see, the Vanderbilt samples produced no tumors. The Platy talc, just the background level of no experimental significance.

But note the middle column. The Vanderbilt sample contained some very long, thin fibers like the asbestos samples. Those fibers are not the elongated, affable cleavage fragments common in this talc. Those are too short and too fat. These fibers are talc fibers. They are relatively rare, but they are observable in Vanderbilt talc.

According to Stanton's hypothesis, this sample should have yielded at least a 60 percent tumor rate, but no tumors were produced. Some have reasoned that the Vanderbilt talc didn't produce a carcinogenic response because there are too few of these fibers in the talc.

In the past we responded to that with, well, maybe. But it is what it is. Still, it is an important question as it does speak to broader fiber risk issues and theories. So we did have a cell study conducted with a
concentrate of these fibers to be tested against an equal amount of asbestos.

(Slide presentation.)

MR. KELSE: Another slide you can hardly read. The results of that comparison study is reflected here. The talc concentrate sample acted differently than the asbestos sample on appropriate cell cultures, which happened to be rodent tracheal epithelial and pleural mesothelial cells. 

Again, suggesting that more dimension is likely involved in fiber toxicity.

I might add that these fibers described as academic curiosities are not easy to find in the air samples. Also, although not pertinent to MSHA, this talc is used in paints and ceramics primarily. The particulate in this talc is bound in the matrix of these products. Thus, there is very, very little, if any, public exposure to this material, unlike vermiculite or platy talc used in talcum powder.

These are the results of a second animal study by William Smith of Fairleigh Dickinson University. Dr. Smith also tested Vanderbilt talc against asbestos. He even took a concentrate of the non-asbestos form, tremolite, prevalent in the talc and tested that against tremolite asbestos, the real thing.

The results were the same as Stanton, tumors for
asbestos, no tumors for Vanderbilt talc and no tumors for
the tremolite cleavage fragments. The mineral component in
this talc most often confused with asbestos.

Knowing that the situation in Libby, in part, promoted the MSHA rulemaking, it should be noted that Dr. Stanton tested the vermiculite mine in Libby. The Libby samples produced tumors comparable to the asbestos samples, while the Vanderbilt samples produced no tumors. That data is not on this table, and unfortunately, was not published by Smith. However, it is now public record that a sample of the vermiculite was provided to Smith, and that he actually got as many tumors with the vermiculite as he did with the asbestos samples.

Before I switch gears and move to non-malignant respiratory disease, I'm well aware that several cases of mesothelioma are said by some to be linked to Vanderbilt talc. I'm always at a loss as to what to say about that because I'm not aware of any mesothelioma cases that have been reasonably linked to this talc. I use the qualifying term "reasonably linked" because we do know that such cases have been reported.

Two were reported, in fact, in the mortality studies. In the cases we are aware of, either the diagnosis was questioned when further investigated, or the latency was
far too short to implicate Vanderbilt talc, or there is a work history of actual asbestos exposure.

When this issue was raised during the OSHA rulemaking in the early '90s, we found that most cases reported never worked at Vanderbilt talc. One case in the most recent mortality study, for example, involved a man who worked for two-weeks in 1948 as a surveyor at the Vanderbilt site with little, if any, talc exposure.

This man then went into the oil business and tore oil burners out of homes during the '50s and '60s. In another case only a 15 year latency elapsed from the first exposure to Vanderbilt talc and death. The latency period didn't fit. In a more recent case the second pathologist found the case unlikely to be mesothelioma after reviewing the tissue and disease process involved.

Before we could accept that such a risk is linked to this talc, we would want the diagnosis confirmed because it is not an easy diagnosis to make. We would also want to confirm that the cases are actually linked Vanderbilt talc, and we would want to know about other possible exposures. I don't think these expectations are unreasonable.

I should also point out that the animal studies, not the cell study we just discussed, are pleural injection and implantation studies. Animal studies of this sort are
typically viewed as having more to do with pleural tumor induction or mesothelioma risk than they do with lung cancer. In these studies Vanderbilt talc did not produce pleural tumors while asbestos while under the same conditions did.

While I don't think this is a factor here, I also want to point out that many older mining facilities do contain real asbestos. Our own talc facility, Vanderbilt, which was built in the late 1940's is no exception. I found asbestos-containing installation on boilers, steam lines and dryers. I've seen asbestos-containing brake linings used on stuffer machines, asbestos-containing floor tiles. Even the use of asbestos as a filtering aid in the mine laboratory.

Much of this has been removed, encapsulated or otherwise replaced with non-asbestos material. But it is important to understand the pervasiveness in older plants. Something that has nothing to do with the ore itself.

This brings me to non-malignant respiratory disease and the question, do we see a lot of dust-linked lung disease suggesting that asbestos, or something just as bad is present. As with the cancer experience, we actually know a great deal about the pulmonary status of our miners.

Radiographs are routinely obtained and date back to the opening of the mine in 1948. Over the years, they've
been reviewed by many pulmonary specialists. Pulmonary testing is also routinely conducted. A very experienced occupational dust disease pulmonary physician and a former director at NIOSH has reviewed the chest x-rays and pulmonary function tests of all our talc workers every two years for the last 18 years.

I think this statement by Dr. Palick, now at the University of North Carolina School of Medicine, pretty much cuts to the chase. Please note, if you can read this, that Dr. Palick does not feel he is dealing with an asbestos-like dust risk. Note that he finds very, very little in the way of pneumoconiosis among these talc workers, and very little progression when some evidence of dust involvement is observed.

In fact, at the end of 1999, note, he finds only one worker with evidence of pneumoconiosis. Our most recent surveillance effort, which we just completed, shows the same results. Remember, this assessment is from someone who has actually looked at these talc workers over an extended period of time. It reflects actual observation.

Frankly, I believe our pulmonary experience with dust is among the best in the mining industry, not the worst. Dust disease is certainly possible with over-exposure to Vanderbilt talc, just as it is with durable dust.

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mineral particulate of a respirable size of any dust. Certainly some dust like asbestos, or crystalline silica pose an elevated risk because less exposure is needed to result in harm.

It's important, however, not to improperly attribute one dust risk to another simply because some level of risk exist for both. When we do see evidence of interstitial scarring, parenchymal opacities consistent with pneumoconiosis, it has almost always been among miners who had, had previous exposure in other area talc mines now no longer operated. Smoking has almost always been involved as well. We do tend to hire miners with prior experience. It's a double-edged sword, unfortunately.

(Slide presentation.)

MR. KELSE: This slide underscores the important of dose or exposure level. You've heard some testimony this morning about coming home covered in white and your car is covered in white, and I don't doubt that for a second. When you compare the dust exposure associated only with the Vanderbilt mine to the dust exposures associated with other area talc mines, you can see why miners exposed to these much higher dust levels might well show dust-linked problems. Happily, such exposure no longer exists. And these are exposure levels that go back into the '50s and

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'60s. It's not yesterday.

The Vanderbilt dust levels have to do with the use of wet drilling and a variety of mill dust controls not present in these other mines. Not so modern or innovative today, but certainly in the '50s and '60s, it was a radical improvement over mining practices at the time.

One x-ray finding that some people fail to differentiate, and wrongly link to asbestos exposure in this talc mine are pleural plaques. The fact that exposure to all talc, including cosmetic talc can result in pleural plaque and thickening is not understood by some physicians who link this only to asbestos.

Plaques are typically seen after 10 or 15 years of exposure in asbestos mines and well as in talc mines. We do see this in our talc workers as well in about 4 to 6 percent of our group. It's important to understand, although this is one condition all talc exposure share with asbestos.

Pleural plaques are not pre-malignant lesions. Clinically, they are reported to have nothing to do with the evolution of mesothelioma or lung cancer. That's a different biologic process with different end points.

These pleural effects are merely a marker of exposure to talc, or asbestos, and likely other dust as well. As this table reflects the pleural abnormalities that
we do see in our talc workers are not associated with pneumoconiosis or pulmonary restriction; although, pronounced pleural thickening can affect pulmonary function. We don't have any one with pleural thickening. We have seen a couple of cases in the past, although it was relatively rare. This underscores the distinction between this pleural abnormality and actual impairment.

In regard to pulmonary function specifically, we do see a thoroughly high prevalence of mild to moderate obstructive pulmonary impairment with very little or no radiographic evidence of an underlying dust involvement. I think it's pretty clear here that our experience here is most closely linked to the elevated smoking prevalence that I mentioned earlier.

The amount of smoking that persists among these miners does bother me. We do offer smoking cessation assistance. We don't get many takers, unfortunately.

I'm very glad that our miners and millers are among the most studied in the world. I'm glad we've conducted the type of medical surveillance that we have, and happy that so many mineral scientists, health researchers and physicians support us and stand behind us.

There are a lot of lessons to be learned from this seemingly endless saga. This, I believe, are among the most

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important. Substances should always be called by their proper name and regulated on the basis of reasonably demonstrated risks. We need more clarity in our exposure descriptions, not less. To do less, I believe, actually compromises worker protection because it obscures our ability to accurately identify cause/effect associations and properly attribute current and future risks.

When the word "asbestos" is thrown about loosely, the very survival of a company, people's jobs can be put at risk when this emotionally charged word is used. It is important that it be used properly. Prudence to err on the side of safety is a good thing. Unbridled prudence, however, can produce witch hunts. Good science is critical if we wish to minimize bias and control the diversion of limited resources to lower-level risks.

I want to say that there is no question in my mind that over-exposure to Vanderbilt talc, International talc, Loomis talc, anybody's talc or just about any durable respiral particulate can cause problems. We've seen it. There is no question.

The fact that this talc in this region is a very complex mineral blend. That it is understandable that people confuse it does not mean that you can attribute, make assumptions or do circular reason. Well, it contains

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asbestos so, therefore, it's got to be an asbestos risk.
Okay, it doesn't contain asbestos, but it seems like we have
an asbestos risk so, therefore, it's as bad as. So you keep
going in this circle that never ends. That's why every
single time -- every time there's a federal -- the record is
opened by any federal agency to discuss asbestos in any way,
shape or form, it seems Vanderbilt is at the table.

It seems like the door is open and right away,
everyone rushes in to talk about definitions and changes,
and maybe they should be considered. But I think you need
to call substances what they are. If you have a fiber that
works or acts just as bad as asbestos does, you need to put
that on the PEL table and say "treat as asbestos," but you
don't call it something it isn't.

The fibrous actinilitie is as bad as tremolite
asbestos is, you should regulate fibrous actinilitie as
severely as you regulate asbestos because it's been
demonstrated to be just as bad. But you don't get a whole
category, or a group, or blob things together because I
think that more than fiber dimension is involved. I think
psycho-chemical properties have a link to this. Nobody
knows what the actual mechanism of asbestos path in the
genisicity is.

A lot of asbestos workers who die of lung cancer
also happen to be smokers. So it's not surprising on our

1 table just about everybody was, if not everyone was, but

2 that's not unique. That's also seen among asbestos workers.

3 It could be that these fibers, because they act almost like

4 magnets, attracts some of the carcinogens and cigarette

5 smoke hold the particles, the particles go to the air

6 exchange region of the lung and then are broken down,

7 encapsulated, digested, produce active oxygen radicals,

8 produce cellular diversities that ultimately end in

9 aberrations that end in cancer.

10 Nobody is absolutely sure, but that's all the more

11 reason why every single exposure that you look at you need

12 to very carefully characterize that exposure. It doesn't

13 mean it's an excuse, or a reason not to regulate or control

14 it. But it's not an excuse to develop sweeping definitions

15 and drag all sorts of things in that there's evidence that

16 they don't act the same way.

17 That's the reason why I felt compelled to come to

18 this hearing so that it is clear what we know about the

19 experience of our miners and millers. I don't know what the

20 experience with Loomis Talc was. I don't know what the

21 experience at International Talc was. Are some of the areas

22 that we mined similar to those areas? Yes, they are.

23 Are some of the mines that were operating in the

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'40s, '50s and '60s still operating? No, they're not. So you have to look at it today. Even if you believe that the dust caused excess lung cancer among the miners, the underground mine was closed in 1995. I don't know really what more to say about that.

I do have slides that do discuss the mineralogy that shows the difference between cleavage fragments and asbestos. It shows talc fibers, and things of that nature. I didn't plan on using those because it's really not pertinent to the five questions that MSHA asked. But if you have an interest in seeing those, and getting an idea of the distinction, you know, what's the difference between these minerals -- what do these terms mean?

I suspect you're to hear more of that probably from the crushed stone industry would be very adamant about not being inclusive of cleavage fragments, for example. You'll probably hear that in Virginia.

MR. PETRIE: It would be up to you whether you want to present those into the record.

MR. KELSE: I think I'll probably hold off because I suspect that, that's going to be a major presentation in Virginia. I think they'll probably be some mineralogists that are going to be prepared to sit there and talk about this 3 to 1, longer than 5 business and how you probably...
need to look at things at a much higher aspect ratio, and
look for populations and stuff that -- particles that were
actually closer to the actual dimensions of real asbestos
and use that as a screening method before you go to sublight
work and spend a lot of money and time.

If you can't see dimensions like that under light
acrosophy, you probably don't have an asbestos environment.
So if you did see that type of population, then you'd want
to take it to the next step, and you'd want to get it
analyzed thoroughly with -- would sublight work.

That's it. To the best of our knowledge, that is
the health experience of Vanderbilt talc miners, past and
present.

MR. PETRIE: The slides that you have shown this
morning, will you be able to provide us with copies of those
for the record.

MR. KELSE: Yes, they're in the folder.

MR. PETRIE: They're in here? Mr. Kelse also
presented several documents for the record. I would just
like to go through and read the title of those documents
into the record. I'll do that at this point.

The first one is just entitled "Public Comments;
the second one is, Mortality Among Workers at a Talc Mining
and Milling Facility; the third is, A Nested Case Control
Study of Lung Cancer Among New York Talc Miners; next is,
Similarities in Lung Cancer and Respiratory Disease Mortality of Vermont and New York State Talc Workers; next is, Relation of Particle Dimension to Carcinogens and Affable Asbestos and other Fibrous Minerals; next is a Reanalysis of the Stanton et al. Pleural Sarcoma Data.

The next one is, Biologic Test of Tremolite in Hamster; next is Mineralogical Features Associated with Cytotoxicity and Proliferative Effects of Fibrous Talc and Asbestos on Rodent Tracheal Affable and Pleural Mesothelia Cells.

The next one doesn't have a title per say, but it's dated 11/29/02. It has was I presume is the name of the author, Brian Boehlecke, MD., MSPH. The next one is a letter dated July 6, 1995 to Dr. Morgan from a Dr. Garcia. The next one is a submittal to an OSHA docket by our R.T. Vanderbilt Company. The docket is H-033D.

The next document is, The Regulatory and Mineralogical Definitions of Asbestos and their impact on Amphibole Dust Analysis. The next document is, the Asbestiform and Nonasbestiform Form Mineral Growth Habit and their Relation to Cancer Studies. And lastly, Asbestos, health risks, and tremolitic talc, the never-ending Saga.

Thank you, Mr. Kelse.

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