**Q. Let us turn now, Professor Gordon, to the particular case at hand. Will you first give us sort of a general description of the investigative work which you have done with respect to the subject matter of this particular case?**

A. Yes. I believe my interest was aesthetic at first, in the gases being emitted by Hoerner Waldorf, and occurred in 1962 when I was spending lots of time along the Clark Fork River in the area past Frenchtown and going toward Alberton, and also in the early sixties I spent a lot of time collecting needle cast fungi in this area, and hunting and so on, and noticed that there was quite a bit of necrosis in the area which I did not observe in other areas, such as Bass Creek, where I was also collecting. However, I didn't bother with it too much because, as I said earlier, I was much more interested in needle cast fungi, because at this particular time I thought fungi was the most serious pathogen to coniferous forests.

Anyway, my interest continued and I took pictures of the area and some of the vegetation in the area. Took pictures of plume coming out and how it laid along the valley there at various heights during inversion periods.

In about 1965 I started to collect vegetation which, you might say, in somewhat of a scientific manner; not keeping anything more than maybe picking up five or six needles, bringing them in and doing histological studies. . . .

And studied and found out that I had a very unique type of histological damage through the photosynthetic cells in the plants as well as to the vascular tissue and to the epithelial cells within the resin canals. And then I later on, when I was comparing this work with Garrison and with previous work that Dr. Solberg had done around—or with hydrogen fluoride under controlled conditions at Pullman, along with the materials that I was studying from Columbia Falls, I started to put a pattern together on the disease syndrome, the histological disease syndrome. And at various times I have talked to individuals from the plant—Bill Hodges, for instance, and we were often asked to give talks together. Sometimes we would; sometimes we wouldn't. And I was at that time claiming that there was damage being done as early as 1964, 1965, somewhere in there, that there was damage being done by the toxic gases being emitted from the Hoerner Waldorf.

I continued to travel through the area to see, you know, what the extent of this damage was, macroscopically, and then went —this was, I would say, 1967—pardon me. 1966, 1967—and bringing branches in then with completed needles on them, seeing if there was a difference in the growth of the various years of the needles on the conifers, and so on, and extending the areas that I had traveled on the surrounding slopes of the Missoula Valley Regional Ecosystem, and had pretty much an idea of where the extent of the visible damage was occurring. . . . I would show this, I would use a two-by-two slide of photomicrographs of the tissues involved, and I would show healthy tissue being damaged by the toxic gases from Hoerner Waldorf; tissues being damaged under controlled conditions within the lab; tissues being destroyed in the field around Columbia Falls; tissues which were destroyed by the hydrogen fluoride in Garrison, Montana. What we did is just make a comparison. . . .

**Q. Well, let me ask again: Did you make any report or presentation to any Montana State agency with respect to this?**

**Comment:**

Whenever possible, counsel should see to it that the information and data on which the litigation is based has already been

furnished to federal and/or state pollution control agencies and that the litigation arises because they have been unwilling or unable to do anything to improve the conditions in the regional airshed. The issue of "exhaustion of administrative remedies" is often met by this type of testimony.

A. Yes. And in my final report to them I sent slides of the—pardon me—I sent colored photographs to Health, Education and Welfare in my report of damaged conifer needles from around Hoerner Waldorf.

**Q. Has there been any response from any state or federal agency to your report, insofar as it relates to this case?**

A. Prior to the action, even before my really getting very serious about the action, I called Mr. Ben Wake—whom I consider a friend of mine—and asked him if it would embarrass his department, the state board of health, if an action was brought against Hoerner Waldorf. He said the prerogative was yours.

I also, right at the time of filing or just prior or just after—I can't remember—I called William McDonald, who is head counsel now for the abatement branch of health, education and welfare. He took Borcher's place, remember, the man I talked to before who invited me into Garrison. We had correspondence before and we had talked on the phone several times—William McDonald and I. . . .

**Comment:**

Make sure that your principal scientists touch base on a regular basis with the appropriate pollution control officials involved in the local region.

**Q. As far as you know does the state board of health, or any other state agency, or the HEW department or any of its agencies have any active program under way at the present time with relation to the pulp mill situation here in Missoula?**

PLAINTIFF'S COUNSEL: I am going to object to the question unless it can be shown that the Department of Health, Education and Welfare, and the air pollution branch thereof, has any active programs anywhere.

DEFENDANT'S COUNSEL: They have one in Maryland, don't they, counsel?

PLAINTIFF'S COUNSEL: If you can show it to me. They might.

**Q. I would like to ask you to describe, without too much complex professional detail, the differences between the several sulphur compounds that are mentioned in the complaint? And these are hydrogen sulfide, methyl mercaptan, dimethyl disulfide, and sulphur dioxide. . . . Well, do I understand from this that you think the principal villain in the emissions is hydrogen sulfide?**

A. *Along* with the low carbon mercaptans, low number of carbon mercaptans.

**Q. Are you able to discriminate between the hydrogen sulfide and the mercaptan as to which is the more serious?**

A. Not at this time, no....

**Q. Do these various sulphur compounds, in your opinion, all combine to cause one single kind of damage to the plant's life? Is this your opinon?**

A. You would have to define the single type of damage. The primary damages to the photosynthesis in the area, the reduction of photosynthesis in the conifers, but you bring in many aspects to demonstrate this reduction in photosynthesis. Now, here you would have to take in necrosis of tissue; you would bring in maybe the lack, the reduction in chlorophyll pigment; bring in the stoppage of the metabolic cycles, maybe by attacking the Kreb cycles, something like this. So what you are doing I can't say. The major damage is the photosynthetic rate of the conifer, by visible damage or invisible damage. This means by what you can observe, where you can actually see necrotic necrosis, or in areas where it is green you don't see necrosis here, there is a reduction in photosynthetic rate due to the emissions of the Kraft mill.

**Q. Maybe it would be helpful to our understanding here if you would define this photosynthetic condition that you speak of.**

A. Photosynthesis is the most important step in the fixation of energy, the only source of energy, the light from the sun, and it takes sunlight, carbon dioxide from the environment. Carbon dioxide passes into the leaf or needle in all green plants, in the vegetated portion of it, and there is a reaction with water and sugar is produced. So, with carbon dioxide, water, and the energy from the sun, produce sugar, and the sugar is the beginning source of energy and it goes many different routes in the plant to bring about the development of tissues, the reproductive cycle, and so on.

**Q. In other words this is just the main stream of life of the plant?**

A. This is the most important part of all life, because man depends upon this process.

**Q. Well, now, how does the pulp mill emission affect this?**

A. It reduces the amount of photosynthesis occurring in the vegetation in the area.

**Q. And how does that get accomplished?**

A. By causing necrosis of the needle, therefore you have reduction in the amount of area carrying on photosynthesis.

Also, what I am working with, there is an increase because the emissions are there, the toxic sulphur compounds are there, they accumulate in the conifer tissues, and as the increase of the sulphur goes up, and this is not a normal situation, and pathologists call it a diseased situation, if it is reducing the photosynthetic rate of the plant, of the tissues involved.

**Q. Well, now, this sounds to me like you are saying that the plant doesn't grow as much as it would otherwise—**

A. That is true. . . . Now, as you can see, a reduction in needle size from year to year in many cases in the conifers in the area, like a dwarfism—we had a dwarfism in the Garrison, Montana, area—same thing, you affect the thriftiness of the organism and you get a reduction in growth and reduction in metabolism.

**Q. And how do you identify the sulphur compound as being the cause of this?**

A. I personally identify them two ways: I identify them with the disease syndrome that I am getting in the tissues, the macroscopic symptoms; the microscopic symptoms, and the accumulation of total sulphur; the data I am getting back from WARF (Wisconsin Alumni Research Foundation); the total sulphur accumulating in the vegetation around Hoerner Waldorf, and as you increase the distance away from the pulp mill you decrease the amount of total sulphur being accumulated in the vegetation.

**Q. Well, is it the fact that you find an increased accumulation of sulphur that you feel, identify the pulp mill emissions with this condition?**

A. Yes.

**Q. What information do you have as to the rate or extent of this increased accumulation? . . .**

A. The data from WARF, and continuous investigation, sending more materials in. . . .

**Q. Now will you describe how you take these samples? Can you tell by any visual observation whether a tree is affected or not, and whether it is one that you want to take a sample from or not?**

A. Well, we are interested not only in visible damage but invisible damage, so we are taking—the Doug fir we took where there was no necrosis, no necrotic tissue on the Doug fir. Now, the last samples I sent in I think every one of them had some ne-

crosis, but the overall tree had necrosis of various degrees on the needles. But you have to make sure you take the whole needle and don't just get the section that is necrotic; you have to take the whole needle from the fascicle to the tip of the needle, and once in a while you take six—have to explain this a little bit: See, each year the apical meristem, the terminal buds elongate, and in that elongation new needles are released, and this usually occurs in June in this area around here. About June 15 our ponderosa pine break and this occurs and you get a whole new growth that has never been exposed, because it had been protected by this terminal sheath.

**Comment:**

Living plants are perhaps the most effective air pollution monitoring system known to man. They are able to accumulate quantities of atmospheric pollutants and act as a kind of summation device which can be read at any time to determine the total amount of pollutants accumulated in a particular area over a definite period of time. This type of testimony from a plant pathologist makes up for the lack of monitoring records over long periods by federal or state health or pollution control agencies.

It also provides a check on chemical or physical monitoring equipment now in use, since the plant monitors are more sensitive and more stable than most man-made devices.

**Q. Just never been there?**

A. No, it is there, but it is hidden and very well protected in a case of armor; it can withstand everything, just about everything—toxic gases, sometimes no. Anyway, each year you have this, if you have a needle down here (indicates) and you can tell by the terminal bud scars, when this terminal bud opens it leaves a scar behind it so you can count back how many years a particular needle has been on the tree. Now, if I wanted to find out how much sulphur has accumulated from 1966 until now all I have to do is go back and tear off those needles produced in June of 1966. See, trees are tremendous monitoring systems of air pollution. This is one thing that the engineers are even starting to agree, that pathologists were right all along, that vegetation is some of the best monitoring systems that you can have today. So, anyway, I can go back and get the conifer needles and take them off from 1967 and 1968, and this last batch I sent in, this is one thing I did, I took needles produced in 1968, from 1968 until now, needles produced June of 1967 until now and separated them and put them in separate packages and sent them in. This way we will know the difference between how much sulphur accumulated between 1967 and 1968.

**Q. Does there appear any difference out in the woods between**

**one tree and another, and the effect of these emissions on them, or is it uniform over the whole area?**

A. No, no. As you drive into Garrison, I think it is the best example to use, you can see dead trees ten miles as you go about from Gold Creek all the way to Garrison, but there are many trees that are alive; there are many trees that are damaged. There is a difference in degree as to susceptibility and resistance to toxic gases.

**Q. Is that true also of the effect of sulphur on the trees?**

A. It is true of all living systems. There is diversity within each species. Within a species the individual will react separately to the causal agents.

**Q. Well, then, if—we will say over across the Clark Fork, to the west of the mill—you are gathering samples of pine needles, or fir needles, and here appears to be a healthy tree and here is one where there are symptoms of necroses; if you took the needles from both and sent them in for this same kind of analysis, would there be a difference in the amount of sulphur accumulated in them?**

A. Very little.

**Q. The healthy one would have about as much as the unhealthy one?**

A. They are both diseased.

**Q. Well, will you explain what the disease is, then, and how it manifests itself one time in a necrosis and reduced growth and in another not?**

A. You can look at a tree that is necrotic and you can see definite necrosis, like it had been burnt. Look at the trees in the same stand, such as in Kramer's yard, and you will have various degrees of resistance and susceptibility. Right in Kramer's trees there are two or three stumps of trees that had died in the last two years. As you go back across those twelve trees in that yard you will see a complete degradation from completely susceptible, which are the dead ones, to ones that are slightly resistant to ones that are fairly resistant. All of them have some necrosis on their needles. I thing it is tree number nine that has very little necrosis. And this is a variation within this particular area with this particular clone of damage.

**Q. Well, what I am trying to ask, I guess, is: Whether one tree has a capacity to exclude the sulphur compound while the other takes it in, or whether the difference lies in the fact that it endures it without apparent change in condition?**

**Comment:**

When using natural systems, such as plants or animals, as monitors of the effects of air pollution, the differential resistance of individual species and individual plants or animals within a species must be accounted for, just as the individual differences among the reactions of human beings who might be exposed to the same pollutant but react differently must be explained. The witness must be ready to answer this question in detail or the record will be worthless.

A. As far as visible; but invisible, no. We are getting—I can't talk for Dr. Sheridan, but his work is showing that you get a reduction in photosynthesis. You have a tremendous reduction of photosynthesis not related to necrosis. Invisible damage, damage you can't see is occurring throughout that area because there is a reduction of photosynthetic rate in that area compared to the controlled areas.

**Q. I am sorry to be so dumb, but I don't see how, what appears to be a healthy tree can be diseased when along side it is one where the disease is manifest.**

A. It is quite easy, if I could explain it this way: I look maybe like a very healthy man, but I could be dying from cancer from the inside. Only a pathologist or a diagnostician may tell you this. While I look very healthy a specialist has to tell me that I am sick.

**Q. Well, I grant that, but there is usually some inner-symptom that is detectable about the one who is afflicted with the malignancy. I guess maybe I should ask you how you tell an apparently healthy tree that is nevertheless sick with this reduction in photosynthesis? . . .**

A. The symptoms in the photosynthetic rate, a pathologist doesn't look at the symptoms of the metabolism that is occurring here, what you are looking at is the fact that metabolism is being reduced or has stopped, and so you look at the end product, or the product in between.

Now, the plant physiologist can tell me, or tell anyone, how much the photosynthetic rate has slowed down. I can tell you when it has stopped, or that there is an abnormality in a number of pigments within the particular cell which would cause a reduction in the photosynthetic rate of that particular plant.

**Q. Maybe what we are after here is actually just a very brief discussion on the pathology, what pathology is and the disease syndrome of the coniferous trees; is that what—**

A. As the causal agent enters through the epidermal area it

comes through a little opening called the stomatal opening, past the hypodermal. On a conifer needle, what you have is the epidermis and hypodermis, be all the way around, and the photosynthetic areas here, which we call mesophyll tissues, and in these mesophyll tissues of the organelles, like the nuclei and the chloroplast, and all these sort of things—and golgi bodies we're not worried about right now—and inside of these things you have another protective layer called an endodermis, and within that endodermis is a transfusion area; this is just for holding water, and then you have two vascular bundles made up of phloem and xylem tissues. This is sort of a pipeline where food is transported down to the root and water brought up from the roots. Then on the outside, depending on the species, and sometimes depending on locality, is called resin canals, and inside of these resin canals we have secretory cells, and then just within the secretory cells we have epithelial cells.

This is what makes a conifer such a good monitor, because it has so many tissues in comparison with a broad leaf plant; so this is why we use conifer.

As the gas comes in, and ideally it would be carbon dioxide, because with the carbon dioxide and the water that comes up from the roots, and the sunlight, as soon as the water gets into the cell, and the carbon dioxide gets into the cell, which it does, it passes in through the cell, but not always as carbon dioxide. Carbon, water, sunlight and chlorophyll pigment, you have the development of sugar I talked about.

If a toxic gas comes in, such as toxic sulphur compound or hydrogen fluoride, it can replace carbon dioxide. The first symptom would be a varying symptom only a pathologist would be able to recognize. Then you can take the two and you can see there is a reduction in carbon dioxide when toxic gas is introduced.

So the first symptoms will be somewhat of a breakdown somewhere of either the pigments within the cell, or maybe the gas itself will attack the nucleus, and since the nucleus is the controlling body the metabolism is not normal within that cell. The gas doesn't stop there, it can also go out to these resin canals.

A lot of these cells are thick and some are very, very thin; have various types of cells, and the thicker cells we call more of a sclerenchyma type; and very thin cells, the exchange of gases and water is much easier than in the thicker cells. So these epithelial cells pick up the gas, too, and these cells in this particular case around Hoerner Waldorf, the cells start to undergo hypertrophy and hyperplasia. What it does, these cells

start to enlarge, and what happens you get an occluded resin canal. What used to be a pipeline, it is now occluded and solid. This is what you might call a cancerous type growth.

We will forget that area and come back and go through the endodermis. The endodermis will undergo collapse. It doesn't undergo atrophy, one-half of it collapses. The reason why only one-half collapses, one side of the cell is very thick walled, this is on the side of the mesophyll; it is thin walled on the inner side towards the transfusion tissue. This causes a breakdown, and now we are in the transfusion tissue, and within this transfusion tissue there are thick walled cells and many thin walled cells, and now the toxic gas does not affect those thick wall cells, but what it does it causes hypertrophy again of the thin wall cells. So you undergo this cell enlargement, and finally it gets larger and just collapses, just like blowing up something that shouldn't be blown up, and finally just collapsed like blown too high.

Then it comes down and continues and it gets into the phloem, into this little pipe system, the one that took the food down to the root system and to the other parts of the plant; this is called the phloem, and the underneath side is the xylem, made up of the thick walls, and the phloem thin walls, some thick walls in the phloem, but the thick walls here do not undergo this hypertrophy, but the thin ones do undergo this cancerous type growth indispersibly, these are inter-dispersed among thick wall cells, and as they enlarge they dislocate the thick walled cells. Anyway, they are displacing them and lose continuity with each other, no more translocation of food out of this particular area. And so with this you have a collapse of the cells.

During all this time the plant actually can be, in that area where this is happening, can be green; a very light green, to be sure. It is not necrotic yet. What it is, what we call chlorotic, starting to become yellow. Symptomologists go on and have millions of terms, like doctors have millions of terms for symptoms, but be the same type of condition, however, and this is what is occurring here. The whole process, in many cases, reduces the photosynthetic rate, because if you destroy, I mean disrupt even this water system as it comes in, then the water will not be present to build that sugar, and also there will be the fact that the sugar is not moving out because the phloem is destroyed, and so on. So, the whole process—

**Q. Just unhealthy and limited in its effect. Now, how does this have an impact on the tree? Does it grow less, or change colors, or what?**

A. It does grow less. We have less growth, as far as needles are

concerned. There is a graduate student—doing annual growth, the reduction of annual growth in the area. I have not seen his data at this time. . . . He told me he is going—I don't know how many cores he has; several hundred cores he has, so on. So it is a very safe statement to make that if there is a reduction, if there is a disease you will have an impairment of the metabolic processes within the plant, and this is what we call a disease, it is a diseased condition, and you have a plant that will not survive. We don't know how long it will survive. This is unfortunate. We can't say this tree is going to die ten years from now. It is just like a doctor trying to diagnose a patient who is a terminal patient, he can't tell exactly when the patient is going to die.

**Q. Well, now, is this loss of photosynthesis a new discovery?**

A. No. I think this was apparent in Don Adams' and Solberg's papers when they did the work with hydrogen fluoride under controlled conditions and the conditions that I have just talked to you about, the whole pathological condition, toxic effects, the affect of toxic gases, of hydrogen fluoride upon conifer needles, or the emissions from Hoerner Waldorf. There is a difference in the degree and the way of the path of the whole disease syndrome between the sulphur and the hydrogen fluoride.

The major difference between hydrogen fluoride and the sulphur compound from the mill will be at the time that the gas, the first point would be the time that the gases affect the particular tissues involved. Now, you have to know that the symptoms that I am describing are between the necrotic area and the green area, and usually they are right in closer to the green area than they are to the necrotic area. I get a needle here and one-half of it is dead; I don't do any histological work on the dead tissue, you can't find anything, and you have to take the tissue that is still just barely surviving, or just undergoing the first effects of the toxic gases; so we get out a very, very small piece out of the middle here, maybe two millimeters long, and this we process through the histological method and look at it, and we find the symptoms in that, what we call transition zone. Now that tells you where I took the tissue from, and you do this both with hydrogen fluoride.

Sulphur emissions from a Kraft mill, you have a—well, I will start with the epithelial cells in the resin canals. The differences between fluorides and sulphur emissions here would be a thicker wall after the enlargement of the epithelial cells caused by the sulphur compounds, you get thicker walled cells, or it appears thicker walled by several magnitudes than with the hydrogen fluoride. It is a slower process of hypertrophy

than you get with hydrogen fluoride gas—it immediately collapses. With the sulphur emissions it is a slow process and a very—well, a thickened type of epithelial appearing cell.

In the case of the transfusion tissues, between these two vascular bundles there is quite a bit of parenchyma tissues, and these are affected very early with fluorides, and they collapse you always have a cavity here. With sulphur emissions it occurs late in the same area. In the fluoride it is just the opposite; it occurs much earlier and it is way in the green zone and the collapse occurs long before—I shouldn't say long—a millimeter before it gets to the dark zone—I mean to the necrotic zone. There are two parts of this phloem tissue here; one which we call active and the other inactive, and the reason they call them this is because the phloem cells closest to the xylem cell, the ones transporting the water, are more active in transporting food than the ones closest to the transfusion tissues. With fluoride the phloem tissues closest to the xylem cells collapse early. With toxic sulphur emissions you have the collapse of this area which you call the inactive phloem first. So it just reverses itself with these two. There is a difference in stain in the transfusion cells as they undergo enlargement and then collapse. Using identical stains, identical staining technique, the stain is taken up, and the stain should be purple to red, it becomes very orange when the needle is being damaged by sulphur emissions. In the fluoride case it has a very red appearance in comparison to the orange caused by the sulphur emissions....

**Q. Do all of the sulphur compounds that are described in the complaint and in the answers have the same effect in the photosynthesis process?**

**You sort of speak of sulphur compound as a group name, and I want to know if it just refers to all of these and they all work the same.**

A. If I use a gas, a single gas under controlled conditions in the lab and I inject a single gas by itself, like hydrogen sulfide or sulphur dioxide, I do not get the same symptom, by itself, that I get from the composite emissions....

**Q. What difference, if any, did you find between the impact of the sulphur dioxide and the hydrogen sulfide on needles when you used them in the laboratory? Is there any difference?**

A. Yes, there is. Remember now we go to the resin canals, and with epithelial cells it takes—usually there is not the occlusion, you don't get the occlusion, the cell collapses while they enlarge maybe three or four times—I shouldn't say three—I should say two to three times their normal enlargement. In most cases,

small exceptions—not many—there is a collapse of these cells before the canal becomes completely occluded; this is one. Wherein, sulphur dioxide, it does flow, always shows a complete occlusion before the collapse of the cells. That is one difference between them....

**Q. I mean, is hydrogen sulfide a quicker acting or slower acting chemical than sulphur dioxide?**

A. Oh, it is slower than sulphur dioxide—...

Then we go into the transfusion tissues, and with hydrogen sulfide alone—I can say this in general throughout the thing, you do not get the tremendous increase with hydrogen sulfide by itself as you do with sulphur dioxide by itself. I would say it is a one-to-two ratio.

**Q. Now would you describe the process by which you expose a needle to the gas of the sulphur dioxide and the hydrogen sulfide?**

A. The exposure is done in a plastic—I should say a fiberglass chamber which has an inlet and an outlet. It has a small fan within this to move the gases continuously. It has a platform and a bracket to hold it tightly down so that the gases do not leak from this particular chamber.

The volume of the chamber is known and given quantities of gases are titrated into the chamber.

The plants, before they are put into the chamber, are coated with—using hundred fifty-two degree melting paraffin, pouring it out on a plate and letting it get a little cooler than that so there is a solid coating between the inside of the pot and the tree so there would be—what I am doing this for so there will be no reaction between the sulphur compound and the soil within the pot here, and then also plug the bottom up with wax, too, so there is no reaction of the sulphur with the water. The sulphur dioxide is very reactive with water. Then it goes in here you titrate your concentration. The concentrations that I have been working with are rather high. We start with the sulphur compound at fifty parts per million to the volume within the thing; ten parts and twenty parts per million within the chamber of the particular gas, either hydrogen sulfide or the sulphur dioxide....

With sulphur dioxide we are getting necrosis within twenty-four hours at around ten parts per million—I think it is ten parts per million—and with hydrogen sulfide I believe it was—it has been a long time, I would have to go back to—I think it is somewhere in the neighborhood of thirty parts per million or forty parts per million....

**Q. Now, you didn't tell us for how long a time these plants are exposed to this?**

A. Usually I will leave them in there until I see the necrosis appearing, because what I am after is, as I said, the transition zone so I can duplicate, see if there is a duplication of the disease syndrome within that area; so I have to have the transition zone. You could show loss in chlorophyll pigment, things like that, but that is not what I am interested in. I am interested in the whole disease syndrome in the conifer needle....

I think the longest we have run our experiments—I can think back—is about four or five days.

**Comment:**

At this point counsel for the defendant attempted to compel the witness to produce the raw data sheets on the experiments then under way at the laboratory. Dr. Gordon refused to release the data on the grounds that it was essential to the preparation of publications by certain graduate students for whom Dr. Gordon was responsible. Such publications were necessary to complete the requirements for their graduate degrees. The issue was whether unpublished raw data compiled for purposes other than litigation in the regular course of educational activities by a witness were discoverable. It is the environmental advocate's duty to protect the future of any scientist and not sacrifice the entire educational effort of a young scientist or at least a number of years' work for the sake of a particular lawsuit, without the consent of the individual concerned, in this case, the graduate student, not just the senior professor. It is also the duty of the senior professor to protect the student, as demonstrated in this case by Dr. Gordon.

PLAINTIFF'S COUNSEL: Now, for the record, I would like to ask Dr. Gordon two questions before we produce any information.

BY PLAINTIFF'S COUNSEL:

**Q. Doctor, is that material that you refer to, the data that you have back at your office, was that prepared for scientific publication or for presentation in this trial?**

A. It was prepared for scientific publication.

**Q. Is that material ready to publish at this time?**

A. No, it is not.

PLAINTIFF'S COUNSEL: All right, I object to the disclosure of any of that material until it is ready for publication.

DEFENDANT'S COUNSEL: I propose to ask the questions when we resume, and if they are not answered I will take it up with the Court.

PLAINTIFF'S COUNSEL: That is fine. You can question him as much as you like about his opinion and the work in progress, but the data is not going to be available, since it is not the work product of any expert for this particular lawsuit, unless it is ready for publication in scientific form.

There is such a thing as scientific and professional ethics involved in these. . . .