BY DEFENDANT'S COUNSEL:

Q. Just prior to the noon recess I was asking you about some laboratory tests that you had conducted artificially to expose plants to some of these sulphur chemicals; is that right?

A. Yes, that is true.

Q. And you said you had some results at the office that would give you a more precise recollection of how the tests were conducted, right?

A. Yes.

Q. Do you have those with you?

A. No, I do not.

Q. I say: To you, as a scientist, were these sufficiently alike, the materials and vegetation growing around the Hoerner Waldorf Plant, to give you a valid comparison from your test results?

A. Yes.

Q. And from the result of those tests have you formed a part of your opinion as to the effect of the emissions of the pulp mill on the Missoula Ecosystem?

A. Yes.

PLAINTIFF'S COUNSEL: I am going to object unless, at this point, it is clarified whether this is a portion of his opinion that has already been presented or a portion that is to be presented.

BY DEFENDANT'S COUNSEL:

Q. Well, will you explain why you did not bring the results of these tests?

A. Yes, I will.

PLAINTIFF'S COUNSEL: I am going to object unless we first specify what portion of this witness' opinion depends upon those test results, if any. . . .

Comment:

The purpose of this objection was to protect the record, and the witness, from possible contempt proceedings.

A. First and foremost this is raw data, it has been collected over a matter of three years, long before there was any thought of litigation against this particular mill. I did this work because I was interested in it, and I should say my research assistant and some of my graduate students helped me to do this work. We put this data together just for the idea of scientific research.

I have never, nor do I know of any scientist who would release a mass of raw data prior to having it in a publication ready to present, or have it already accepted by a national journal. If it did happen that I were to release raw data there is a good possibility—say I released raw data to another scientist whom I did not know very well; just gave him the raw data, maybe that he would publish it. I don't mind losing the publication, I don't think it is very important, because I have a national and international reputation as a mycologist and as a plant pathologist. However, it does hurt the chances of the graduate student publishing the paper to rise in the academic world, or even in the environment of industry.

Q. Well, do you expect to utilize any of this information in testifying in this case when it comes to trial?

PLAINTIFF'S COUNSEL: I am going to object. It calls for speculation on the part of the witness and refers to something that isn't—an event that has not yet been determinable. We don't know when this case is going to come to trial.

A. I do not know, actually, if I am going to use any of this data. I know we are going to publish it, however, and when it is ready for publication I will send a copy to other environmental scientists, and if you would like a copy of it I would send you a copy of it also....

Q. Then these simulated exposures that you made in the laboratory are included as a part of your overall collection of histological specimens that you considered in making up your opinion about the toxicity of the Hoerner Waldorf emissions?

A. This is true, yes.

Q. And I take it that the raw data that you speak of as being in your possession includes the data as to the manner in which the material was exposed in the laboratory to the gases as well as the histological slides that you got from the plant after exposure?

A. That is true.

Q. And you do have that information but you choose not to reveal it because of the explanation you make?

A. Scientifically unethical to reveal it....

Q. You used the term sub-lethal quantity or—didn't you—sub-lethal level?

A. I should have used the word sub-necrotic.

Q. Or whatever you call it. Can you express that in any kind of tangible quantity?

A. At this time my studies are, as I said previously, to show histological damage and the concentrations that I have been using, as I told you previously also, are data that I will not release.

Q. Well, the answer is that if you have an opinion you will not disclose it?

A. No, opinions I will disclose. The fact—

Q. —not any raw data that you are going to publish and transform into any other kind of data; just the opinion.

A. Yes, I have an opinion that hydrogen sulfide causes histological damage.

Q. I wasn't talking about that. I was asking you about quantities.

A. Lethal quantities cause necrosis to tissues.

Q. Can you express this in parts per million per hour or per day, or per any other period?

A. That would be the raw data. That is not an opinion, that is my raw data which, I said, I will not release, because scientists —I never heard a scientist being asked, ever, to release raw data that is not in publishable form so someone else can get it and use it in a scientific publication... I can give you an opinion on the effects of sulphur in the chamber and in comparison to the effects around the Hoerner Waldorf Plant.... My opinion is, from my work carried out in the lab, it is damaging to conifer, conifers grown in this toxic atmosphere. And I can also categorically state that sulphur, to the best of my knowledge, and all of the work that I have done, that the sulphur emissions from the pulp mill cause, are toxic to the conifers growing in the Missoula Valley Regional Ecosystem.

Q. Do you base that in part upon these tests that you conducted in the laboratory that you are discussing?

A. I base that upon the fact that I have the largest collection of diseased conifer needles probably in the world. I don't know of anyone who has a larger collection.

I base it upon the fact that I probably have done more work on histological study on toxic gases than anyone I can find in the literature, or that I have ever talked to.

And I base it upon a long association with observing dam-

aged conifers in areas where toxic gases are being emitted. . . . Well, basically I compare it with all of the diseased syndromes that I know of, and so I automatically compare them. You compare them with a healthy one and you compare them with diseased ones, so on. One attacked by fungi, attacked by frost, those died of natural attrition. . . .

Comment:

The witness has now established alternative bases for his opinion other than the unpublished data, protecting his testimony from a motion to strike as an opinion unsupported by any evidence.

Q. Now, does this photosynthetic injury that you have described cause the necrosis of the needles in the pine and the fir?

A. I thought I explained that earlier. It is not photosynthetic injury, it is—as a pathologist I have never heard the term used before. . . .

The loss in food production causes a weakening of the tissue and eventually the tissue will succumb and necrosis does occur, yes.

Q. What I would like to have you give us your opinion on is: whether these five or six types of plant injury that you answered in the interrogatories, all are results or follow from this photosynthetic reduction? . . .

A. Necrosis of needles, accumulation of sulphur, pollen abortion, presence of mercaptans and shifts of conifer terpenes, lessened growth.

Q. I got the impression, however, from your testimony this morning, that all of these consequences tend to flow from the decrease in the photosynthetic rate because of the way it interferes with the plant's life and function. All I am asking: whether this is a correct understanding or not?

A. Maybe I can explain it this way—I don't know if I should explain it in that particular manner—well, anyway, the photosynthesis of a plant is a very important function of that plant, and everything in that plant depends upon the buildup of the food, the growth of the plant, the seed production of the plant, the health of the plant, the ability to ward off attack by fungi and insects all depends upon the metabolism of that plant—the photosynthetic rate; and, therefore, if the photosynthetic rate is reduced there will be—completely destroyed—there will be either no seeds; or partly destroyed—excess seeds in some cases, or absorbed seeds in other cases, so on.

Q. And ditto with needles and growth?

A. Growth.

Q. I don't know how to interpret this one about mercaptans and the shift of terpenes, yet; but I get the impression that this decrease in photosynthesis is the basic health of the plant and these other things follow from it as normal sequelae?

A. I don't mean to make it the most important thing in the world; it probably is, but still you can't take part of the operation away without destroying the whole scheme of life here, and what you have, it starts out this way: The very lowest plants are single cell with green pigment, and that green pigment allows them to survive. But they have other factors, too. They still may have to be able to produce an exoenzyme to get the inorganic material to bring in the cell; and what is more important: the exoenzyme or the green pigment? The green pigment is the first order of magnitude; has to have the food to produce—well, they have to have the sun for the energy and carbon dioxide and the chlorophyll present to produce the food to eventually produce the exoenzyme. So it follows in this order; I guess I will just have to come back and probably say that the photosynthetic rate is—the photosynthesis is a very important function. A function that separates, in many cases, man from animals.

Q. Let me ask you now about the permanent or temporary nature of these disruptions in photosynthesis: What is your opinion on that?

A. Well, unlike animals that can replace damaged tissues, plants cannot replace damaged tissues in this way. For instance, when a plant is hit with an ax and you remove a small piece of it you are going to form there a small canker on the plant, and while it will heal over, the scar will always be there. It is permanent. And when a needle, the tissues in a needle are killed there is nothing to replace them because there is no, what we call meristematic tissue within that needle. So, once the cell is killed there is no replacing it, it is irreversible; it is dead and it never will function again. There is no way for another cell to come in and replace it, to regenerate and replace this. So every bit of damage is permanent. When fungus attacks a plant, if it drops a needle from that tree early, which we call early casting of the needle, that is permanent damage to that particular needle—it is gone.

Q. Well, how does this affect a tree as we see it growing in the woods? Will it grow less high or less big around, or will it just die? At what point does this serious consequence manifest itself?

A. Well, depends upon the sensitivity of the plant itself. Like I mentioned this morning, within a single species—for instance,

within the single species *pinus ponderosa* there is a tremendous degree of susceptibility and resistance. . . .

Q. In other words there is an individual response by each tree to this same exposure?

A. Sure; just like being infected, each and every individual in this room being infected with a cold virus, maybe some be absolutely immune to it and others come down with a severe cold; some with half a cold, so on.

Q. How rapidly does this effect reach a serious proportion in an individual case? Is there a percentage or degree of affectation that you can tell us about? What effect does watering have on it?

A. You can prolong the life of an individual by maybe giving him a particular antibiotic; maybe like they did with General Eisenhower, though he was going to die, and there wasn't any way to save him. They prolonged his life a period of time by careful, loving, tender care. You can do the same thing with a tree that is bound to die.

Q. Well, trees have a life, by the time they reach maturity in this area, of what, a hundred fifty years?

A. No, that is when they cut them; that is the death of the tree, to be sure. But a tree, they forest-cycle-out due to fungi, primarily. But usually if the forest were completely controlled and the cutting of the forest was—if you left the forest to be virgin they will eventually go out by the cause of wood fungi, wood decay; maybe some cases, if you have too close crowding, insects come in and clear part of them out. But the eventual destruction of that particular forest, say you planted the whole forest at one time, would be wood decay, fungi, and you may have some thinning out by insects and other lesser pathogens, and so on. But the annual increment is finally slowed by the rate of decay that comes in and finally, as it cleans out the middle—you have probably seen trees that have a hollow center, been completely destroyed—eventually the tree will topple. The whole forest will go this way in wood decay, the forest, per se. But in the meantime another forest is coming in and taking over, so succession goes on. Maybe a different group of trees; a different species of trees in some cases, too, but the year-cycle in this area has never been worked out. I gave a talk at the forest symposium held on campus this fall and spent quite a bit of time trying to find anything on literature thereof. I did give my talk, finally, after talking to the numerous personnel in the forest service and going down looking at some of their records and talking about the virgin forest in Rock Creek,

so on. I made a rather broad statement saying that the forest, Douglas Fir forests in this area probably cycle about three hundred fifty years, every three hundred fifty years.

Q. Well, the point of this is: Over what percentage of that life-cycle can it be killed by exposure to these sulphur gases?

A. That is very, very difficult to figure out. . . .

Comment:

The defendant based much of its case, just as the industry based much of its propaganda, on the lack of evidence of direct mortality from air pollutants emitted by the plant. Any environmental litigation based on direct mortality as the sole evidence of damage is doomed, and the wise environmental counsel will couch the complaint in terms of general degradation to the regional ecological system, one element of such degradation, perhaps, being necrosis in a particular plant species or individual plants or animals. At every opportunity counsel must object to any attempt by the defendant to establish mortality as the basis of the plaintiff's claim of damage in environmental litigation involving an entire regional system.

PLAINTIFF'S COUNSEL: I am going to object to any further questioning along this line on the grounds of relevancy and materiality.

There are no allegations in this complaint or no statements in the interrogatories that have been already served, that I can see, that indicate that the case is predicated upon actual death or total destruction of any trees in any given area here. I think the line of the complaint is that the presence of such noxious sulphur compounds is demonstrated by the obvious indications of odor, diminished visibility and damage to plants.

I think this is the extent of damage to plants that should be the subject of inquiry from the witness.

Comment:

In this particular suit, the plant damage was pleaded primarily to permit testimony about plant pathological states as indicators of general air quality levels.

Q. Now, how long does a needle stay on a tree naturally?

A. Naturally it varies. In the forest, you know, just like on the forest service land where I am collecting, and some private land, so on, where the tree is not watered like in a yard or something like that, the normal retention for a needle is three and a half years, and I am basing that half year on the fact that they break in June and they will fall in the fall.

You know in the fall a lot of people think the trees are looking very badly because the fourth year needles are falling, being cast at that time, so it does give the forest a kind of brownish

appearance, even though it is an evergreen forest in ponderosa pine.

Now, Douglas fir you go longer periods. Go six or seven years before natural attrition casts them from a healthy tree.

Q. What about the larch, it casts its needles every year; does it get affected by these sulphur gases?

A. I have walked on a few of the ridges there that have larch on them and I have not collected larch to bring it in. There is a problem in this particular case that I don't—I like something that I can use for the whole year around and left larch alone. I have observed larch, like I have taken pictures of damaged larch in the Columbia Falls area, but I still don't do histological work on them. Besides, the tissues in the larch are rather bad to work with; they are not as exciting as ponderosa pine.

Q. Now will you discuss the difference between younger and older pine and fir trees in their susceptibility to the sulphur gases?

A. I have not done any controlled planting of trees around Hoerner Waldorf. We are expecting to put out some plots maybe this spring. But the fact is if you get a young tree, a fast growing tree, you get a little more damage. This is an opinion, and I base it not only on what I observed around Hoerner Waldorf, Columbia Falls and Garrison, these places here, but the real vigorous growth, if there is going to be vigorous growth, is due to the environment—a lot of water, lot of nutrients—and more damage on these than on a very slow growing tree.

Q. That has to do with where they are located; but what about the age factor—does an old tree grow less rapidly than a young tree?—Is that what you are saying?

A. As far as elongation of the terminals, I took the terminal bud breaks, and then you get the elongation of this material, and usually the young, fast growing trees will produce more of a leader than an old tree would. The damage, I would suspect would be—it is my opinion of this, of what I have observed in areas where toxic gases occur, that the damage is more severe in trees that grow the fastest. This is typical of most pathological fungi. For instance, the tree is most susceptible to fungal attack during vigorous growth than in slow growth, in many cases.

Q. How do you translate this into young and old? I don't think I understand that yet. . . .

A. All I can say as a pathologist: They are all being damaged by toxic sulphur compound. That is what I am saying.

Q. As you walk around in the woods you can certainly see the necrotic needles, can't you?

A. Yes.

Q. Do you see them on the young trees or on the older trees or on both?

A. On both.

Q. Would that be some symptom of the effect?

A. No, it is showing me there is a presence of something there that is causing a necrosis of needles and, therefore, if the macroscopic symptoms are somewhat suspected of being caused by toxic gases, might be caused by fungi, insects. I have to determine that in the field. If I can't determine it I bring the branch back to the lab and do the dissecting and go into the process.

Q. But in your cruises around and observations of things can you say whether you see more signs of necrosis of needles on the trees under ten feet high, or on those over ten feet high? Just to pick an arbitrary point.

A. I think it is easier to say that the closer one gets to the mill the more necrosis one sees.

Q. Now, then, let me ask you whether this necrosis takes the form of browning of the entire needle so that it falls off, or only part, so that it remains there, but some green and some brown, or whether it just turns pale or what?

A. It is the whole degradation. You can have a quarter of an inch or you can have an inch on a needle, or you can have three inches. The needle will not cast until, usually—well, I will qualify this in just a minute—the needle, if it were strictly a sulphur compound causing it, the needle would not cast until the tissue dies all the way back to where the abscission layer is normally formed, and as soon as it hits that point the needle is cast.

Q. Is that the little pocket in which the thing gets fastened on to the branch?

A. Yes, it is a fascicle that holds, right. The abscission layer, this is where the needle, my hand (indicates) this was the fascicle (indicates) the abscission layer is going to form right there.

PLAINTIFF'S COUNSEL: Indicating the point of attachment of the fascicle to the bowl?

A. Yes.

Comment:

It is imperative to convert witnesses' gestures into some accurate verbal description for later use. Never permit the mere word (indicating) in the transcript to substitute for a describable activity.

Q. Over how long a period of time will needles die, back until it gets to this point of being cast?

A. How long does it take, is that what you are after?

Q. Yes.

A. In natural attrition—maybe I can use this in natural attrition—it takes a matter of about three months where this is going to occur.

Q. That is when the three and a half year old needle is ready to cast off it will turn brown in about three and a half months?

A. Doesn't turn brown in the way the gas causes it. The whole needle becomes a paler and paler green, and then all of a sudden you have a brown appearance, brown necrotic tissue.... It just falls off.

Now the degree—going back again—will depend upon the susceptibility. It could occur—say you could get an inch on a single dose of the toxic gas if it were, say, a twenty-four hour period that the wind blew continuously toward a particular tree, the concentration was just right coming in there, might get an inch of death on, say, a half-way susceptible tree. Rate them one to five; the fifth being most susceptible; the one being most resistant, and number five you might have three inches of death from that single dose, from the twenty-four hour period. But I am theorizing now, because I have not sat out there for twenty-four hours and watched needles become necrotic before my eyes.

Then in a resistant tree, the same concentration, the same period of time, you may get a quarter inch, and some you may not get any.... that area that is now necrotic is more susceptible, because there is a natural guarding of the tissue. The epidermal layer, and the cutin is laid down by the epidermal cell by little small projections like this (indicates) and the cutin is laid down now in the necrotic needle, that necrotic portion, and it loses the weather—just doesn't have the protection. That cuticle is for protection, and now the insects, the less aggressive insects can now get on there and feed on there. And since they have an opening, now the door is open, they might take the needle.

Q. And crawl down the end inside?

A. They are called leaf miners; you know, like a coal miner.

Q. Now, when the tree is standing here all year long and is evergreen, I take it it is vulnerable to this kind of damage from exposure to the gases any time during the year?

A. Yes.

Q. There isn't any particular difference between summer and winter . . . the fall and the spring . . . as far as the effect of gas on the needle is concerned?

A. Oh, yes, there is. See, when the photosynthetic rate increases in the spring time—especially the spring time—you get the break of the bud and new needles emerge, tremendous metabolism, building the food for the whole thing to go, and this is when most of the damage will occur.

Usually if you had to pick a season where most damage occurs, would be in the spring when the very quick, vigorous growth is occurring, because this is when the high rate of metabolism, including photosynthesis, occurs.

Q. The mature needles, the one year, two year, three year ones, they don't grow any more, do they?

A. No.

Q. Do they kind of change inside and become more active in the spring time, is that what . . . ?

A. No. Actually, if I were to have a branch, or tree with branches of three years' needles on them, and I take this, and the fourth year, it is in June, and we are just going to take the growth now and introduce a toxic gas in the environment, most of the damage to those various aged needles will occur on the current year needles. The other needles are not as susceptible because they don't have the rate, the photosynthetic rate as the current year needles. This is really a vigorous growing part of it, and this is really where the action is occurring, is where the gases and the nutrients accumulate.

Q. Well, then, how long does it take for this new growth to reach to full size?

A. Full size? About a month and a half.

Q. Does it continue to be more sensitive than the one, two, and three year needles?

A. For that whole season, yes; the growing season. If there is any damage, for instance, on the 1968 needles, you know that that damage, right now if I went out and collected all of the 1968 needles, all the damage that is there has occurred from June of last year, middle of June of last year until the present time.

The damage that is occurring on the 1967 needles I wouldn't

have known unless I had measured them in 1967, because they are continuously exposed and they might have picked up another inch of two inches of necrosis during that period of time.

Q. Well, in general, the closer to the source of emission the more serious the damage? Or is there any such correlation?

A. This has to do—if there is an answer, that it could be correct, it takes in a lot of factors. The topographical factors of an area; the climatological factors and how high the stack; there is a temperature inversion occurring, and so on. So you can say that there is—in fact it is quite easy to say that there is more damage at a particular level above the, along the side of the mountains, than there are many cases down at the level of the river. But, now, this is certain localities, and so on; yet the river would be closer than up to the side of the slope to the source.

Q. There are just lots of variables, then, in this situation?

A. Yes, there are.

Q. Well, will you explain what are the mechanics of the effect of the sulphur compound on the tree? This is a gas, you have said, not a substance that is deposited on the needle, is that correct? And as I understood you this morning, in effect the plant kind of inhales that gas which it gets along with the carbon dioxide?

A. Yes. The opening cannot discriminate between a gas which is sulphur and a carbon dioxide.

Q. In your opinion does a given pine tree have accumulative tolerance above which it is going to show harm, and it doesn't make any difference whether it gets this accumulation in one exposure or in a year's continuous exposure?

A. You will have accumulated effects regardless if it is a single shot or over a long period of time. . . .

Q. Then you do not have any opinion at this time as to the sublethal level for hydrogen sulfide?

A. I have an opinion that hydrogen sulfide is a toxic gas at various concentrations. The concentrations depend on many factors: The susceptibility, resistance of the tree and the meteorological conditions. What I really have an opinion on is that hydrogen sulfide is a toxic gas to vegetation.

Q. I guess nobody disputes that, do they?

A. No.

Q. The question is concentration, isn't it?

A. Yes, sir.

Q. And the degree and extent of exposure?

A. Yes.

Q. Okay. What constitutes a lethal quantity then?

PLAINTIFF'S COUNSEL: I must object again. The question has already been answered: As much as is present where that tissue damage is observable in the Missoula Regional Ecosystem.

A. You want me to answer that?

DEFENDANT'S COUNSEL: Yes.
(Reporter reads pending question to the witness.)

A. That which takes to cause such things as hypertrophy of the epithelial cells in the resin canals and breakdown of the mesophyll cells, the reduction in chloroplast, the hypertrophy of the parenchyma cells of the phloem tissues and of companion cells. . . .

Comment:
The witness should always be encouraged to answer the question as precisely as possible particularly when it is asked by opposing counsel.

A. Well, you can have a frost, a late frost in the spring. Say we have nice, warm weather like this for, oh, a month, and the Doug fir breaks now instead of breaking when it should at the end of April, then all this young foliage starts here, and then all of a sudden we get a real cold snap there will be, can be a tremendous amount of necrosis. Sometimes can occur on a particular area of a mountain where you get a real cold snap. This gives you, reduces the photosynthetic rate by a long shot and causes a tremendous amount of browning in the area. Frost is one of them.

Q. Rainfall, moisture, the lack thereof?

A. Yes, drought can cause a necrosis, and it is an interesting pattern; the pattern is: the old needles fall at first, then you have four year needles fall; then the three year needles fall; the two year needles fall. The younger needles are more susceptible. It depends on the time of year. If they are not out you will hit the older needle, so on. But you will get damage to the younger needle. Versus, drought comes from the back door and frost from the front door.

Q. And hot conditions, hot, dry periods; does that have any effect?

A. It can. When you get a very warm wind, and for some physiological reason you don't get the stomatal closing enough, transpiration, excess transpiration during this hot period, and you can have burning of the needles. We call it burning.

Q. And does that have the same form of necrotic progression as does the kind you have described here with respect to the sulphur compounds?

A. Macroscopically you have to—I don't think you could definitely tell the difference in all cases between drought or frost or boran from a toxic gas, such as the sulphur compounds. There are a few characteristics that are common among the sulphur compounds and hydrogen fluoride which you will not find from the drought, but they are just slight, slight banding, this sort of thing.

Q. Maybe it would be helpful if you would explain the difference between macroscopic and microscopic? I think you have been using both terms here. I am confused in that respect.

A. Macroscopic is something I can observe with my eye without the aid of any glass, magnifying glass, and microscopic is that which I observe through the microscope.

Q. Now, does just extreme cold, a real cold winter make a difference?

A. There has been quite a bit of work done on cold and the effect of cold. Progressively getting cold, you know, at a normal rate, somewhat of a normal rate like we usually have, usually does not affect the conifer needle. In fact even though they are small and thin—in fact sometimes fifteen degrees centigrade difference between the outside of the needle, on the surface of the needle and within the center of the needle. Most of this work that I have been talking about now has been done up in Canada; even colder areas than we have. But you can have some damage, winter damage from not only cold, but maybe a wind burn along with the cold, except for the cold temperatures, so on. But usually the tree, it has been here for literally hundreds of millions of years, and they have learned how to evolve into survival from natural causes.

Q. Well, when they are thus affected and survive—I probably asked you this before, in a way, but I still don't quite understand the situation—they live on in spite of this attack, whatever it may be, many times, don't they? The reduction and the photosynthetic rate, are they kind of permanently crippled to whatever extent this thing affected them once, or do they grow around it and get new health restored by additional years of branches and needles and things?

A. That is right, because we are talking about a cold spot or a drought for one year; real cold period of time, or an insect attack for six years, or something like that. It is not a continuous thing.

Now, if we had thirty below zero all the time our conifers would disappear, the ones we have in this area; but this wouldn't be true. The continuous emission of sulphur compound, which would be continuous, is quite different from a cold or drought period for a short period of time.

Q. Have you seen any signs of any recovery of the pine and fir trees from the low level sulphur exposures?

A. Sure, I can take them out of this atmosphere, under controlled atmosphere, take them out of that atmosphere and put them in the greenhouse and they will survive and put on new foliage. But that dead area will always be dead. But next year they will produce— . . .

Only next year, leave enough green so they can survive next year and produce new foliage.

PLAINTIFF'S COUNSEL: If it will make it any easier for the defense in this case, it is not the intention of the plaintiff at any time to present evidence by direct testimony of any quantities and parts per million of any of these gases that we allege are present as being necrotic, or non-necrotic levels, or sub-necrotic or sub-lethal levels. We consider it irrelevant and immaterial to the subject matter of the action.

DEFENDANT'S COUNSEL: Are you saying, counsel, that your position then is: that any emission at all of this sulphur gas is a basis for the relief you seek in the complaint?

PLAINTIFF'S COUNSEL: No. We are saying that emission which produces histologically significant evidence of damage at a place distant from the actual source of emission of the property owned by Hoerner Waldorf, and immediate vicinity is such as gives rise to and supports our cause of action. And we are claiming, simply, that the level of emissions should be reduced below that level which produces histologically significant evidence of damage in certain indicator plant tissues. That is all the cause of action is.

DEFENDANT'S COUNSEL: Well, what is that level? This is what we are trying to find out.

PLAINTIFF'S COUNSEL: That level is not in terms of parts per million, which is of absolutely no significance. It is the amount that is emitted from those stacks such as to produce within the period of time under observation by Dr. Gordon and other scientists evidence of damage. And we are saying that the obligation of the defendant in this case is to reduce those stack emissions to a level that will not produce such damage off their property; or, in the alternative, proof that it can't be done with the current state of art in pollution control technology.

That is all this law suit is about.

The plaintiff isn't interested in ambient air quantities of hydrogen sulfide, methyl mercaptan or any of the organic sulfides. We only seek that the defendant keep those emissions down to a level where there is no evidence of plant damage, and indicator species, off their property; or, in the alternative, within the state-of-the-art establish that it can't be done.

DEFENDANT'S COUNSEL: Well, it is apparent we are not getting anywhere on this subject, so I will turn to another one.

Comment:

At this point defendant's counsel asked a question for which the witness had apparently been waiting all day. It is again the kind of question you should never ask a hostile expert unless you are sure of the answer and the extent of your own knowledge of the subject matter. It leads irrevocably to further questions that can only further damage your record. In all of the author's environmental litigation, his advice to environmental scientists has been to bury the counsel for the defendant at every opportunity on cross-examination; bury them with facts, and bury them with detail, just remembering that the criteria for acceptability and admissibility of the testimony is relevance, competence and materiality.

BY DEFENDANT'S COUNSEL:

Q. Are there, in your opinion, any other causes for reduction of the photosynthetic rate in fir and pine?

A. Oh, yes, many.

Q. Will you enumerate them, please?

A. Yes, I will.

I will first break it down to the fungi causing a reduction of photosynthetic rate of the trees, and I will start with basidiomycetes which comprise some fifty thousand species, and among these there are at least two thousand which attack Douglas fir and ponderosa pine. The group within the basidiomycetes: we have the rust fungi; the uredinales, as a sub-class title, and within the uredinales we have three families of fungi that have members which attack the Douglas fir and ponderosa pine. And the three families are: Pucciniaceae; cronartiaceae, and melampsoraceae. The cronartiaceae and melampsoraceae have members which attack conifers. The pucciniaceae do not attack conifers.

Now, in the cronartium, which occurs in this area, it is a fungus which causes a strangulation of ponderosa pine. It attacks three other pines: It attacks Jeffrie pine, which doesn't occur in the area; so we won't bother with it. But, anyway,

this particular organism has an alternate host for the causal agent. What they have here is a fungus that goes from one host to the other, produces so many spore stages on the alternate host and so many on the ponderosa pine. The spores that are produced on the ponderosa pine are the aecial stage and the pycnidial stage, and also it produces the uredial stage. This is a five-spored eutypic rust. Now, when the basidiospore travels from bastard toadflax on to the pine it lands on the needles of the pine, travels down the needle into the stem and lives within this area of the living tissues of the stem, such as the cambial region, the phloem region, and just a small portion of the xylem, and also travels down the xylem rays into the pith of the stem. It lives in this area for a short time without any visible host-parasite relationship; which means macroscopically there is no visible damage to the plant.

And then after two years little pustules occur on small swellings of the stem. It can occur on the stem and on the main bole of the tree. If it is a young bole a slight swelling will occur. At this time the pycnidial stage will form. What you have here, this is the spermagonial stage of this particular rust fungus, and it is like a little pustule structure, and from this come long threads which we call receptive hyphae, and within the pustule is produced the female, pardon me, spermatium, and you get the break of the bud, and new needles emerge and a higher than previous rate of metabolism, and one will be plus and the other be minus. So you have pluses and minuses in spermatia and the different pustules. Now, at that time there may be some necrosis of the needles. From the strangulation caused by the swelling of the stem the photosynthetic area of the needle will be affected by the lack of nutrient transport. Then what happens is that a transfer of the spermatia from one pycnial pustule to the other occurs. This is done primarily by insects, because on the outside of the pustule there is a sweet exudate which attracts insects, and these insects transfer this exudate with the spermatia back and forth. A matter of fertilization, actually.

When the fertilization occurs, when the spermatia land on the opposite receptive hyphae the cell wall of the spermatia breaks down and its nucleus migrates into the receptive hyphae, and the nucleus travels down the receptive hyphae into the pustule and then travels beyond—because all these hyphae of this particular pycnidial stage are inter-connected, and finally works its way down into what we call the aecial primordium. This nucleus fertilizes the aecial primordium, and now we have set up the dicaryon stage. One thing I forgot was the basidiospore that infects the tree was monocaryotic, means two nuclei. So

the aecial primordium now can develop. It doesn't develop unless it has been fertilized by this nucleus. Now we develop the dicaryon stage, and at this time the swelling on the stem is increasing. But this is an obligate parasite and it doesn't kill its host. It may kill certain amounts of photosynthetic material on the outside of the stem, but not enough that will destroy itself on this particular stem, because if it killed the stem then it wouldn't be able to survive itself, because it has to have living tissue to survive. Then as this strangulation continues the aecial stage appears and this produces a spore stage which is called the aeciospore stage, and a small eruption to the bark occurs and the spores are released into the environment. These are wind-borne and they travel back to the bastard toadflax which they infect. This occurs in the spring just as the leaves of the bastard toadflax appear. In effect, the cycling of the urediospores and teliospores occur on the leaves of the bastard toadflax. We are not interested in the flax, anyway. This will continue year after year. The pycnidial stage will remain one year ahead of the aecial stage, and over a long period of time, and it is a perennial infection. It can be on that tree for a matter of a hundred years and not cause the death of that tree —this is really what it amounts to. But all during this time it has reduced the photosynthetic rate, this tree is a diseased tree. It will not grow in comparison with a tree not infected by this particular fungus.

There are other similiar rust fungi which do the same thing on ponderosa; such as peridermium.

Q. Do we have these conditions in this area?

A. Yes, we do.

Q. Any insects or other forms of disease that have this effect of reducing the photosynthetic rate?

A. Yes, there are.

Q. What are they?

A. Other fungi.

Q. Without describing all of them in detail can you enumerate them?

A. I have on my desk the host check list of my former professor, C. Gardner Shaw, which is at least two hundred some pages long, with the fungi which attack conifers and other plants in this area. If you wanted me to say there is a tremendous number I definitely will say there is a tremendous number of fungi in the world which attack Douglas fir and ponderosa pine.

Q. Are there any other kind of insects or diseases that do it as well as the fungi?

A. Well, now, diseases are primarily caused by fungi.

Q. I see; all right.

A. And insects cause insect infestation, and so on. Yes, there are insects.

Q. The insects carry the fungi?

A. Some cases they do, yes.

Q. And this is what causes problems?

A. Yes. For instance, this small killing throughout this area on ponderosa pine the last few years, which we call beetle attack, the people were saying was caused by the beetle attack, actually two organisms, a fungus and the dendroctonus beetle, and the dendroctonus ponderosa beetle transfers the ceratocystis organism with it, and the beetle transfers the fungus and the fungus strangles the tree. It is like the Dutch Elm disease organism. It is a kissing cousin of the ceratocystis.

In this area there are insects such as the spruce bud worm, which cause serious defoliation.

Q. You mentioned mistletoe?

A. Mistletoe, it is a parasitic phanerogram. Just an angiosperm, that is all; an organism that I have done embryogenesis on. I have worked on all three species in the area, and it causes severe damage. Quite easy, however, to determine when it is present.

Q. And I think that you mentioned that there are other air-borne chemicals that cause this, too? . . .

—this reduction of photosynthetic rate? . . .

And those are the fluorides? . . .

And what else?

A. I mentioned boron.

This is an interesting chemical. It is not in the air, the highway department puts it on, and they haven't been doing it here for a long time, but it causes symptomatic macroscopic symptoms. The macroscopic symptoms are very similar to microscopic symptoms of gases in the air, and what happens here is that the highway department uses borate to kill, as a herbicide, along the roadside, and they would pour this borate out along the road—keep the weeds down—rather than pouring two-four-D or tordon, one of these; and as it rained the water would wash down the borate in the trees below this. And usu-

ally where you would see they were using this stuff where guardrails are, and the trees below the guardrails became very necrotic and brown. Very similar to a tree being affected with toxic gases. And turned out this was caused by the uptake of borate, an excess of boron.