IN THE UNITED STATES DISTRICT COURT

FOR THE DISTRICT OF COLUMBIA

BETSY LAKIE,)
)
)
Plaintiff,)
) Civil Action
-vs-) No.: 93-0561
) (HHG, DAR)
SMITHKLINE BEECHAM, et al.)
)
)
Defendants.)

DEPOSITION OF OTTO WONG, Sc.D., F.A.C.E.

DATE: March 14, 1996

TIME: 1:00 p.m.

LOCATION: SAN FRANCISCO AIRPORT MARRIOTT
1800 Old Bayshore Highway
Suite 5096
Burlingame, California 94010

REPORTED BY: Barbara Friedman

Certified Shorthand Reporter
License Number C-7845

DEPOSITION OF OTTO WONG, Sc.D., F.A.C.E.

A P P E A R A N C E S:

For the Plaintiff: LAW OFFICES OF HARVEY S. WILLIAMS
BY: HARVEY S. WILLIAMS, ESQ.
1019 19th Street, N.W.
Suite 800
Washington, D.C. 20036
(202) 857-0877

For the Defendant: HOWELL, GATELY, WHITNEY & CARTER
BY: DANIEL W. WHITNEY, ESQ.
401 Washington Avenue
Twelth Floor
Towson, Maryland 21204
(410) 583-8000

13 Also Present: Bettina M. Heusch, law clerk

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PULONE & STROMBERG (408) 280-1252

DEPOSITION OF OTTO WONG, Sc.D., F.A.C.E.

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PULONE & STROMBERG (408) 280-1252

DEPOSITION OF OTTO WONG, Sc.D., F.A.C.E.

OTTO WONG, Sc.D., F.A.C.E.,

called as a witness, after having been first duly sworn by the Certified Shorthand Reporter to tell the truth, the whole truth, and nothing but the truth, testified as follows:

EXAMINATION BY MR. WILLIAMS

Q (BY MR. WILLIAMS): Would you state your name and address for the record, please.

A Otto Wong. 181 Second Avenue, Suite 628, San Mateo, California 94401.

Q Is that your work address?

A Yes.

Q Dr. Wong, I'll show you Plaintiff's Exhibit 1.

Did you receive a copy of the amended Notice of Deposition?

A Yes.

Q Were you able to bring the documents requested in the notice?

A Yes.

Q Why don't you tell me what you brought here today.

A I have a stack of documents that Mr. Whitney's

23 office sent me. It is right here.

24 Q Okay.

25 A And then I have a folder of articles that I

1 reference to in my report.

2 And, also, I have one folder with

3 correspondence and so on between me and Mr. Whitney's

4 office.

5 Q Let me take a look at the folder that

6 Mr. Whitney's office sent to you. And in the folder,

7 those are the articles you referenced?

8 A Yes.

9 Q Do you have any articles in there that you did not

10 reference?

11 A Only one additional article that I did not

reference in my paper.

Q What article is that?

A And a few pages from the IARC, the International Agency for Research on Cancer, monograph on benzene, published in 1982.

Q Okay. Anything else would be the articles you already cited in your report, right?

A Yes.

Q Let me have one second.

Other than the materials you brought today, do you have any other documents related to this case that you did not bring?

A No.

MR. WILLIAMS: Plaintiff's Exhibit 1.

(Whereupon, Plaintiff's Exhibit No. 1 is marked for identification.)

MR. WILLIAMS: Let me show you Plaintiff's Exhibit 2.

(Whereupon, Plaintiff's Exhibit No. 2 is marked for identification.)

Q (BY MR. WILLIAMS): Can you identify that document?

A The report that I wrote dated September 12, 1995 in this case. And also attached to that is my CV.

Q Is the CV current?

A I might have a couple more publications since then.

I did bring a current copy of my current CV.

Q Oh, you did? Okay.

A And this has additional publications. I think maybe just two or three.

MR. WILLIAMS: Let's add this as Exhibit 3.

(Whereupon, Plaintiff's Exhibit No. 3 is marked for identification.)

Q (BY MR. WILLIAMS): I want to ask you some

22 questions about your training. Your degree is in
23 physics and mathematics, that was your undergraduate
24 degree?
25 A Yes.

1 Q Then you studied epidemiology in private school,
2 correct?
3 A Well, actually, I also have a graduate degree in
4 physics, and I switched to public health in 1972.
5 Q Okay. Did you receive a master's degree in
6 physics?
7 A Yes.
8 Q You received that in '72. And then did you
9 actually receive a degree in public health?
10 A The copy -- I'm at a disadvantage. The copy you

gave me is missing a few pages.

Q Let me show you Exhibit 3 which is the most recent one anyhow, correct?

A What I got twenty years ago and today has changed.

I received my master's degree in physics at Carnegie-Mellon University in 1972.

Q Okay. And then following that you went into epidemiology, is that --

A I think I started at the Graduate School of Public Health in either August or September of 1972.

Q Okay. And you subsequently went on to finish -- once you finished the public health degree, then you went to epidemiology?

A No. Epidemiology is part of the Public Health School at the University. So I started -- you can say

that I started taking classes in epidemiology in 1972.

Q Okay. In your CV you indicate that you are board certified in epidemiology?

A Yes.

Q What does that mean?

A The certification body is the American College of Epidemiology. They have two groups of membership. One is what we call Member. You have to have an advanced degree in epidemiology or a related field, and then you have to pass through a couple exams.

And the other type of membership is Fellow. To be a Fellow you have to satisfy all the requirements of regular member, and on top of that you also have to have what they call significant contribution to the Science of Epidemiology.

And in 1982 I sent in my CV and my qualifications and so on to a committee, and they admitted me as a Fellow.

Q Is that what the F.A.C.E. refers to in your title?

A Yes. Fellow of American College of Epidemiology.

Q Now, where is this committee that you sent this

22 to; is that something that's in California?

23 A It is not in California. The correspondence at

24 that time actually was at John Hopkins University, but

25 the officers of the College changed every few years and

1 it depends who has the office. That's where you write

2 to.

3 Q Okay. So you write to the committee and the

4 criteria is having an advanced degree in epidemiology?

5 A Or a related field.

6 Q And the advanced degree, would that be a master's?

7 A I don't remember all the details now, but you got

8 to have a doctoral degree, either a Ph.D. in

9 epidemiology or Ph.D. in a related field, or you can

10 have an M.D. degree plus an advanced graduate degree

that can be either an M.S., master level degree or Ph.D. in epidemiology.

Q Okay. On your resume it doesn't indicate that you are a Ph.D. in epidemiology?

A I have what is called a doctoral degree in science, doctoral in science which is the same as a Ph.D.

Q Does this committee consider that to be the same?

A Oh, absolutely.

Q With that doctorate of science, would that automatically qualify you to be a member of the American College of Epidemiology?

A No.

Q Not a fellow, but a member?

A Not even a member. You have to take some exams in

order to become a member.

Q So to become a member you take a test?

A Yes.

Q And the test is administered by this committee?

A Yes.

Q Is this a board that is recognized by the American Medical Association?

A That I don't know.

Q After you become a member, do you then -- then I take it you submitted some of your work, examples of your studies to the committee in order to become a fellow; is that the way it worked?

A No. The American College of Epidemiology started in, I think, in 1981, so there were a couple hundred Fellows at that time who were what we call "grandfathered" into the college. If you can show or demonstrate that you satisfy the requirements plus you have made a significant contribution to the scientific committee in terms of publications and so on, then they can grandfather you into the college. And I was one.

Q You were one of the grandfathered members?

A Yes.

Q Or fellows?

A Fellows.

Q So this board began in 1981?

A The college started, I believe, in 1981.

Q And when were you grandfathered in?

A 1982. I submitted my application sometime in 1981, and I received notice from them in 1982.

Q And the notice indicated that you were admitted; is that correct?

A Yes.

Q When did you take the test?

A Grandfather means you don't have to take the test.

Q So you didn't have to take a test. You applied and submitted evidence of your studies?

A Right. At that time there were two ways to get into the college. One is you can take the test, or if you think you have already met the requirement and also have made significant contribution, you can ask the committee to look at your CV and see whether they can grandfather you in. And if you fail that, you can always go back and take a test.

Q What is epidemiology?

A Epidemiology is the branch of medical sciences that deals with the distribution and determinants of disease in human population. So there are two components in the definition. One is distribution. And by that we mean which group or subgroup in the general population have a higher rate of certain disease that we

are interested in. We want to identify the high risk group.

And once we have identified the high risk group, we want to find out the determinants of that, meaning the risk factors associated with the high rate of disease in that group.

Q Does epidemiology also include application of information that you learned to alleviate health problems?

A I'm not sure I understand that question.

Q Well, you indicated that you studied the distribution of disease in a group. And you identify risk factors. Do you use that knowledge, is part of the definition of epidemiology to use that knowledge to help to alleviate health problems?

A Absolutely. The relationship between, for example, cigarette smoking and lung cancer was discovered through epidemiologic studies. And certainly we have been using that information to persuade people not to smoke.

Q So would that be a third component of epidemiology in addition to the first two?
A In a sense you can call that.
Q Epidemiology doesn't study the biological or pathological mechanisms by which disease develops in an

individual, does it?
A Yes and no because epidemiology really is very useful in the sense that it doesn't matter what kind of parameters you look at. As long as you set up the right study design, then we can call it epidemiology.
You can use cancer as the health endpoint or you can use some biomarkers as the endpoint. And by setting up the right biomarkers to study, you can get an understanding of the biological mechanism of a disease

process.

Q But you don't -- epidemiology doesn't study how disease develops from a biological or pathological point of view in an individual, does it?

A I think it does in the sense that if you define your endpoint in such a way that you can call that a biological -- well, certainly you can set up your study in such a way that you can study any biological mechanistic hypothesis.

Q But aren't you studying the incidence of a particular endpoint in a population rather than the way that that endpoint develops from a biological or physiological point of view in that individual?

A That's true. In that sense it is not a study of the mechanism, but you would certainly -- epidemiology can be used to either refute or confirm hypotheses

suggested by biological experiments.

Q You don't have any other board certification other than the one we discussed in epidemiology, do you?

A No.

Q So you are not trained in medicine?

A No, I am not.

Q And you are not trained in toxicology?

A No, I am not. Well, when you say when I'm "trained" in something, I took courses in toxicology when I was in school. It is a little bit different from the previous question when you say are you board certified in other disciplines.

Q You don't hold yourself out as a toxicologist?

A No, I don't.

Q And you don't hold yourself out as a physiologist?

A No.

Q Or as a biochemist?

A No.

Q I take it you are not going to offer an opinion in

this case, then, on the biological or pathological mechanism of Mrs. Lakie's disease?

A No.

Q In general terms, tell me, in with causation, isn't it true that epidemiology can determine whether a particular agent is capable of causing disease; is that

correct?

A Yes.

Q An epidemiologist doesn't, in a general sense, embrace determining whether a particular individual's disease has been caused by a particular agent, does it?

A I think it does for chronic diseases.

Q You think that -- explain that if you would.

A One of the -- there are many problems associated

with the determination or causation in chronic diseases.

By chronic disease, I mean, there is a long latent period between exposure to the disease and the appearance of the disease, usually in terms of years or even decades.

And most chronic diseases have more than one etiologic agent. So when you look at a single case, you would not be able to tell what that individual has been exposed to. And you cannot distinguish biologically or clinically that that case was caused by Substance A as opposed to Substance B. In other words, just looking at a leukemia you cannot tell.

If we don't have the employment history or the exposure history, you cannot tell whether that leukemia case was caused by iodizing radiation or benzene exposure or simply a background case. So in that sense you have to rely on epidemiologic studies to

determine the probability of causation due to each exposure factor.

Q So in the absence of a case history, an epidemiologist would be used to establish the probability of causation based on epidemiological studies; is that what you are saying?

A No, what I'm saying is if you do not know the exposure or employment history of a certain patient, clinically, you would not be able to say anything about a causation because just by looking at the disease itself you cannot tell whether the disease was caused by A or caused by B.

Epidemiology is useful in the sense that if you do know the employment history, we can assign a probability of causation to each individual exposure factor.

Q If you know the employment history?

A Yes.

Q I'll take it, then, if there was a case control

study of an individual or case, or clinical study of an individual, if the person's medical history, employment history was known, the clinician or medical doctor would be able to also make a diagnosis as to the -- to render an opinion as to causation of a disease; isn't that true?

A No, he cannot.

Q If the medical doctor knows the patient's prior exposures and he diagnosed a disease, the doctor cannot offer an opinion as to the causation?

MR. WHITNEY: Objection. Speculation.

THE WITNESS: When you use the term "doctor," I assume you use a clinician as opposed to a epidemiologist because the doctor can be a clinician and

epidemiologist at the same time.

Q (BY MR. WILLIAMS): I mean a clinician.

A A clinician cannot make any statements on causation just by looking at that case without any knowledge of the epidemiologic literature on the disease and exposure that person has.

Q How was leukemia first discovered to be caused by benzene?

MR. WHITNEY: Objection.

THE WITNESS: It started with some case reports, but based on case reports we don't have a firm conclusion at that time, okay? The association was confirmed by epidemiologic studies.

Q (BY MR. WILLIAMS): But before the epidemiologic studies, it was through case studies that the medical community became aware that benzene caused leukemia, wasn't it?

A Yes.

Q When you do an epidemiological study, what is required to make sure the research methods are trustworthy?

A I guess the most important thing would be to define what group you want to study, what exposure you want to study, and once you have defined your group, then you have tried your very best to obtain a group that's unbiased, that would fit your definition.

Q Anything else?

A Once you have the group, then I assume you have to obtain some information on the health outcome you are interested in. And again, you have to get complete information on that.

Q Do you start typically with a particular question that you are trying to resolve?

A Absolutely.

Q Do you call that a research question?

A I would call that a hypothesis.

Q For example, if you wanted to study whether benzene causes leukemia, that would be your hypothesis?

A That would be the hypothesis, but the way that you just stated it would be a very nonspecific, general question.

Q How would you state it if you were to embark on

such a study?

A If somebody comes to me and say, "I want to sponsor a study to find out the relationship between benzene and leukemia," let's say, then immediately I would come up with at least two or three questions to ask you. One would be what kind of exposure range we are interested in because by just saying "benzene" is

not sufficient in terms of exposure. Are we talking about very trivial exposure? Are we talking about very heavy exposure?

And you say "leukemia." I need to know are you interested in leukemias of all cell types or specific types of leukemia.

Q If the answer to the first question was you were looking at low-dose exposure, what would your next step be in designing a study?

A Well, I would want to know how low. When you say "low dose," I don't know what that means.

Q Okay. Why don't you know what that means? Is there not a generally accepted standard for what low dose is?

A Certainly, when you talk about occupational exposure, low may mean less than one ppm. But if you are talking about environmental exposure, one ppm may not be low compared to what we come across on a daily

basis.

So when you say "low," I really would like to know what range you are talking about. That would direct me to the total population that I would like to study, which group I would like to study.

Q How about if it was one part per million?

A One part per million, as one ppm, then I would say some of the workers in refineries. At this point, nowadays, I think, they are exposed to less than one part per million, and would be the group that I want to look at.

Q Would the level of exposure that you are looking at also determine size of the sample?

A Once you know which exposure range you are interested in, then you try to find some populations that would fit into that criteria. And sometimes there are lots of people out there that falls into that range, and you can have a meaningful study. And sometimes you

just can't, you cannot find a group large enough to provide you with any meaningful data.

Q You mentioned bias earlier. What is bias in a research method?

A Technically, the definition of bias is a systematic error that would produce a biased result.

Q Can you give me an example?

A Okay. Let's try this one and see if -- let's say there is a proposal to raise the tax rate in high income brackets, and we want to find out what percentage of the population is either for or against that proposal. So we need to talk to a lot of people, include a sample of people to get that information.

And one way to talk to a lot of people or at

least run into a lot of people is, I guess, going down to the shopping mall. So we go down to the shopping mall, set up a table. Let's say we set up a table in front of Neiman-Marcus, and we try to persuade people to give us an opinion on the question as they go into Neiman-Marcus. And let's say they eventually would get a hundred people who would agree to answer the question, and it turns out that 95 percent of them, 95 out of 100, said they are against raising the tax rate.

Now, I know I'm not supposed to ask a question, but let me try to ask anyway. Would you think that the 95 percent obtained through the sample is valid, representative of entire population?

Q What would you think?

A No. The answer is no because it is a biased sample. The people that walk into Neiman-Marcus are more likely to come from high income bracket. Certainly, that sample is not representative of the

underlying population.

Q Can you give me an example, using that as an analogy to an epidemiological study where you are studying exposure of workers in an oil refinery?

A I can give you a more general example than that.

There is something what we call the healthy worker defect, and it refers to the observation that working populations usually have a lower rate of cardiovascular disease than the general population. Okay? And that, basically, is the result of selection bias, people who select themselves into the working population.

In other words, if you have a cardiovascular condition you may not pass through the employment exam and, therefore, you would not be able to work in industry.

So if we look at the employed population,

their cardiovascular disease would usually be lower than the general population. That is the bias.

Q The bias is because the workers you are studying might actually be healthier than the general population?

A In terms of cardiovascular disease.

Q And you refer to a selection bias. Is the bias also attributable to the fact that it may have better access to health care?

A That also may be true. So maintenance of health, that's another component.

Q And would it also be true that part of the healthy worker defect is that sick workers might be removed from the study, from the population that you are actually studying?

A No. That's not true because normally how we define a study -- well, let me give you an example. In most of the refinery studies that we look at, the definition for the study is anyone who works at the refinery more than either six months or a year would be included in the study. And once that worker is included in the study, he or she will be in the study regardless of how long he or she worked.

Q Is that true for all the studies you did, there is a threshold duration of employment that's a requirement to be in the study?

A Usually, there is what we call a minimum employment requirement. And the reason for that is otherwise you would be studying a lot of very short-term workers, maybe a student who work there maybe one or two summers.

Q And it is usually six months?

A Six months or a year.

Q I guess in general these are cohort studies?

A The one that I just described would be a cohort study.

Q And that's to be juxtaposed to a case-controlled study which actually studies individuals and their medical history with regard to exposures to whatever agent might be the subject of the study?

A Well, both cohort and case control studies study exposures. In a cohort study, you start with a cohort of workers exposed to certain chemicals and observe them over time to find out what disease they develop and compare the incidence of disease to some other unexposed group. That would be a cohort study.

A case-controlled study, like you said, is more or less the opposite in terms of time direction. You start with cases of the disease, patients of the disease, and compare their exposure history to some other group, what we call controls, without a disease.

Q Is it true that because of the difference in the approach that case-controlled studies can often detect weaker associations than cohort studies?

A No.

Q They cannot?

A I think you may be referring to the power of the study. I'm not sure what you are getting at.

Q Well, I guess in a general sense I am referring to

the power in that isn't it true that the case-controlled studies are often more powerful than a cohort study because they can detect weaker associations?

A If you are talking about very rare disease, is true that cohort studying may not be practical because you would need a large cohort in order to come up with

just a few cases of that disease.

A case-controlled study would avoid the problem of having a study of tens of thousands of people by concentrating on maybe ten or twenty cases that you can obtain from a hospital.

So in that sense for rare disease, case-controlled study may be more practical. But the disadvantage of case control study is that normally the documentation of exposure is very poor compared to cohort study.

Q If the incidence of a disease is less frequent in the general population, does that require a larger cohort to study a possible association?

A I don't understand the first part of your question.

Q If the incidence of a disease is infrequent in the general population -- let me rephrase it.

Does the size of the cohort, is that inversely proportional to the incidence of the disease

in the population?

A You are absolutely right. If the disease is rare, infrequent, then you need a large cohort in order to come up with some meaning or meaningful results.

Q When you say "rare disease," is there a generally accepted definition of what is a rare disease?

A Well, certainly angiosarcoma or mesotheliomo, both of them would be rare disease. A few cases per million per year. That would be rare.

Leukemia, I don't think is what we call a rare disease.

Q To detect a doubling of disease in a population, if the incidence of that disease was one in a hundred in a general population, what sample size would you need to have to detect a doubling of that disease?

A You are overestimating my ability to be able to do

that right here. There are a couple of formulas that I have to go to and look at. I can't just answer a question like that.

Q Okay. Are these formulas published somewhere?

A Yes. They are all over. Any basic textbook.

Q What textbook would you go to?

A Any textbook that talk about basic statistics.

Q Is this a statistical concept or is it a statistics-combined-with-epidemiology type concept?

A The underlying concept is more statistical than epidemiological because what you are talking about is what sample size should I have in order to detect such a difference. And the difference you just referred to is a twofold difference. It doesn't matter whether it is a

disease that we are talking about or a matter of opinion in the general population. So it is really a statistical issue.

Q Okay. The example I gave, doubling of a disease, would an example of that be if you studied a cohort, let's say, in an oil refinery and found out that there were twice as many leukemias as one would expect in the general population. Would that constitute a doubling of the disease in the population you studied; is that an accurate way to phrase that?

A Yes.

Q And would the relative risk be two?

A Yes.

Q So to define relative risk, you divide the amount, the incidence, in the cohort population with the incidence in the general population?

A Yes.

Q Is there a relative risk that has to be present in order to conclude that the association is statistically significant?

A The sample has to be sufficiently large for you to make the statement whether the result is statistically significant or not. Let me give you an example.

If you have a sample of three, and two out of the three got a disease, okay, so your incidence rate is 66 percent. But that doesn't tell you a whole lot because you only have three people.

On the other hand, if you have 3,000 people and 2,000 of them have that condition, you still have the same incidence rate. But I'm sure you will agree with me, in the second case with the larger sample, you feel a lot more confident about your result than the first one.

Q And with regard to the sample size; that is, whether or not the sample size is sufficient, depends on the incidence of the disease in the general population;

17 is that accurate?

18 A Those two are related.

19 Q So if you have an incidence that is relatively

20 small in the general population, and you study a small

21 cohort, your study is not going to be very useful; is

22 that a fair statement?

23 A By itself it would not be very useful. That's

24 correct.

25 Q Do you know what the incidence of myelodysplastic

1 syndrome is in the general population?

2 A Overall, in the older age group, about 60, I would

3 say I think it is around one per thousand.

4 Q One per 1,000. And that's in the older age group?

5 A I would say -- by "older," I mean above age 60.

Q From age 60 on?

A Right.

Q Do you know what the incidence is below age 60?

A Would be lower. I don't know what that would be.

Q I'm still curious about the formula to determine the sample size. Is there an authoritative text on epidemiology that you would recognize in determining that issue?

A There is a book that I can recall right now that I go to from time to time, and I'm sure that one would have the formula. It is a book by Harvey Checkoway, C-h-e-c-k-o-w-a-y. I think it is called Research Methods in Occupational Epidemiology, published by Oxford University.

Q What other authoritative texts on epidemiology are there?

A The classic, written by a former professor at John Hopkins, Lilienfeld. L-i-l-i-e-n-f-e-l-d.

Q Lilienfeld is the author?

A Yes.

Q Do you know the name?

A I think it is simply called Epidemiology.

Q Are you familiar with clinical trials?

A To some extent, yes.

Q What are they?

A Technically, clinical trials is part of the epidemiology as well. The kind of studies that we just talked about a few minutes ago are more or less observational in the sense that you go to a refinery, you study whatever people did. You have no control over who works at the refinery.

A clinical trial is different. You have some control over which group the patients go into, either placebo group or group that would receive some new medication.

Q What's the purpose of a clinical trial?

A A clinical trial is to determine the safety and effectiveness of some new, for example, medication. You would organize the patients who come to the hospital into either the placebo group or what we call the treatment group, and you observe the two groups over time and determine whether indeed the medication is effective and indeed the medication is safe.

Q Is there a size of the group that's normally tested in these clinical trials?

A The different phases of clinical trial, in the very first phase, Phase 1 or Phase 2, what you are interested in is just to determine what kind of dosage you would give to the patient without any acute

toxicity. So you would want to make the group as small as possible.

Phase 3 is what we call comparative trial. You would have multicenter, larger scale study. And again, depends on what you want to find out.

Q So you don't know how large that third stage would be?

A You use whole -- the same formula to determine the sample size, that the same formula that we use to calculate cohort.

Q Okay. And once a drug is shown to be not causing adverse effects in these clinical trials, it is normally marketed; is that correct?

A I don't know what kind of requirements you got to go through in order to market a product. I don't have that knowledge.

Q Okay. Isn't it true that sometimes after drugs have been shown in clinical trials not to cause any adverse effect that, once they are marketed, they cause adverse effects in some people who eventually either get sick or die? Are you aware of that occurring with

regard to clinical trials?

A I can think of some hypothetical scenario. Clinical trials usually lasts maybe a few months or even a few years. Some of the long-term effects you may or may not be able to observe in a clinical trial.

So I would not be surprised that in some cases you do see some adverse health effect that you did not detect in the clinical trial.

Q So you are referring to a latency period for the adverse effect which was not sufficiently observed possibly in the clinical trial period?

A Depends on the chemical. Depends on the medication. Sometimes it may take longer than the duration of the clinical trial to observe that.

Q Can you think of any other reason why a drug might

cause adverse effects in the general population that were not observed in the clinical trial?

A Well, another scenario I can think of now is the medication, that the drug may interact with something else that becomes more toxic than we study in the clinical trial.

Q Is it also possible that the sample size was not large enough to detect the adverse effects that were later observed in the general population?

A Is possible, but in order to do a clinical trial,

you have to go through the issue of power calculation, power consideration. You have to specify what kind of risk you intend to detect and your sample size is based on that.

So if you conduct a clinical trial based on the protocol, then supposedly, if you don't see any effect with that sample size, that should be conclusive as far as your hypothesis is concerned. You always stay in your protocol, what kind of risk you want to detect in the clinical trial.

Q So I guess the problem can also be with the hypothesis in the clinical trial. If the hypothesis were not correct, then the sample size might not be sufficiently large to allow the adverse effects to be observed.

Is that a fair statement?

A Yes.

Q When were you first contacted in this case?

A July or August 1995.

Q Who contacted you?

A I believe it was Mr. Whitney.

Q Was it by telephone or by correspondence?

A By phone first.

Q Had anyone called you from SmithKline and Beecham prior to Mr. Whitney calling you?

A No.

Q What was the extent of your initial phone conversation?

A I think, basically, Mr. Whitney told me some of the basic facts of the case and asked me whether I would be interested in working with him.

Q Do you recall what he told you?

A Not really.

Q This file here, is this your correspondence?

A Yes.

MR. WILLIAMS: Let's mark this as Exhibit 4.

(Whereupon, Plaintiff's Exhibit No. 4 is marked for identification.)

Q (BY MR. WILLIAMS): Does Exhibit 4 contain all

your billing to date on this case?

A Except for this month.

Q Okay. And all the correspondence from Mr. Whitney is in Exhibit 4, except for transmittal letters in this exhibit which we are going to mark as 5?

A Yes.

Q Is that right?

A Yes.

Q Did you have any other phone conversations?

A After, with Mr. Whitney, after the initial conversation with regard to the facts of the case, we

had several phone conversations.

I cannot tell you today whether we discussed in those subsequent conversations, whether we discussed

the facts of the case or not. I think we spend more time discussing whether the deposition in general would go ahead or not.

Q And those conversations were in January?

A I think it was in January.

Q But you wrote your opinion in this case on September 12th, 1995?

A Right.

Q Has your opinion changed since you wrote your opinion on September 12th, 1995?

A No.

Q You have received records subsequent to September 12th, 1995, haven't you?

A Right.

Q When you rendered your opinion, what was your understanding of the exposure of Ms. Lakie to benzene?

A That she was exposed to benzene through some dental adhesive.

Q You didn't do any calculations, I take it, on the amount of benzene that she was exposed to, did you?

A No, I did not.

Q And when you rendered your opinion in September,

did you know what her total exposure was?

A I rely on some calculations done by Dr. Jacobus.

Q So your opinion is based on what Dr. Jacobus calculated?

A For the exposure part, yes.

Q Does it affect your opinion if you do not consider Dr. Jacobus' calculation, and you assume that Ms. Lakie absorbed 98 percent of the benzene to which she was exposed?

A I should really be more specific.

I say I rely on Dr. Jacobus' calculation. It is not an exact number because all those are, basically, calculated estimates. I really look at the range, what kind of range of exposure we are talking

about, and compare that to what we know in epidemiology.

Q Do you recall what the range of exposure was that you were looking at when you rendered your opinion?

MR. WHITNEY: You are free to look at your report.

THE WITNESS: I remember.

Q (BY MR. WILLIAMS): I think all the pages of your report are here, even if some of your CV was omitted.

A The range was in the neighborhood of 1 to 200 microgram per day.

Q 1 to 200 micrograms per day?

A In that range.

Q That was the range you were considering?

A Yes.

Q Was Mrs. Lakie's disease explained to you by Mr. Whitney?

A He told me what it was.

Q What did he tell you?

A MDS.

Q And I know there are a lot of medical records in here.

Did you have an opportunity to review this in Exhibit 5?

A Yes.

Q What is your understanding of Mrs. Lakie's disease, of what she has?

A It was MDS.

Q By that you mean myelodysplastic syndrome?

A Yes.

Q What is myelodysplastic syndrome?

A It is a disease of the blood and, as well, the bone marrow. I don't know what else you want me to tell you about it.

Q I want you to tell me what your understanding is.

It is a disease of the blood?

25 A As well as the bone marrow.

1 Q In what sense is it a disease of the blood?
2 A It usually affects a number of different
3 circulating elements in the blood in the sense that, for
4 example, white blood cell count would be depressed.
5 Q And would the red blood cell count also be
6 depressed?
7 A In most cases, that would be the case.
8 Q In what sense is it a disease of the bone marrow?
9 A The appearance of bone marrow would not look
10 normal. You cannot function -- it doesn't function as
11 normal bone marrow sometimes.
12 Q Do you know whether or not Mrs. Lakie has any
13 chromosome abnormality?

A I think she did.

Q Do you know which ones?

A 5Q. Actually, she was diagnosed with a very specific type of MDS, 5Q minus.

Q What is 5Q minus?

A 5Q minus simply means that one of the arms of the chromosome five is absent. That's one of the benchmarks of a specific type of MDS.

Q Do you know when the term "MDS" first came into use?

A I believe it was in the early 1980s by the FAB, French American British panel.

Q So prior to the early '80s, the term myelodysplastic syndrome was not in use in the medical

community for this type of disease?

A I don't know whether that term was ever used before 1980s or not. What I said was FAB really come up, developed a classification of the subtypes of MDS and recommend the use of the term "MDS."

So MDS, the use of the term MDS became more popular since the 1980s.

Q Would it be fair to say it became generally accepted, a generally accepted term in the medical community as developed by the FAB group?

A I would agree to that.

Q How does MDS differ from leukemia?

A They are different diseases. Different International Classification of Disease code, certainly, you would treat them differently and so on.

Q Well, does MDS have any relationship to leukemia?

A According to the literature, certain types of MDS have a higher risk of progressing into acute myeloid leukemia.

Q Do you know which types those are?

A I think it is the type that, with what they call excess blast, b-l-a-s-t-s.

25 Q Do you know whether Ms. Lakie has excess blasts or

1 not?

2 A No, she did not.

3 Q Isn't MDS also known as a preleukemia syndrome?

4 A Some people consider those subtypes, yes,

5 preleukemic.

6 Q The subtypes developed by the FAB group?

7 A The type of excess blasts would be considered as

8 preleukemic, yes.

9 Q You would agree, I take it, that benzene is a

10 carcinogen?

11 A I agree.

12 But at the same time, whenever we talk about

13 benzene and we call that a carcinogen, we need to know

what kind of exposure we are talking about because if the exposure is low it is not carcinogenic.

Q Within those parameters, you would agree that benzene is a known human carcinogen?

A Yes.

Q And do you have an opinion on whether benzene is also a mutogent?

A That's outside of my area.

Q You have no opinion on that?

A I have come across papers indicating it is mutogenic, but I have not done any exhaustive research on analysis of the literature, so I won't be able to

offer you an opinion on that subject.

Q Okay. Do you have an opinion on whether benzene

is genotoxic?

A I would give you the same answer.

Q As the one you just gave?

A Right.

Q You came across papers, that is, but you have not done any studies yourself?

A Right.

Q And do you have an opinion whether or not benzene is cytotoxic?

A I will give you the same answer again.

Q You would agree that benzene is toxic to bone marrow, would you not?

A Given enough exposure, yes.

Q How long has it been known that benzene causes bone marrow toxicity in humans?

A I would say there are some case reports going back to at least early part of the century.

Q Do you have an opinion on whether or not benzene causes MDS?

A I think there is some evidence that benzene causes MDS if the exposure is high.

24 Q When you say "high," what do you mean?

25 A As I stated in my report, there are a couple of

1 studies, reports, indicating that when workers were
2 exposed to benzene levels above 100 ppm, they have an
3 increase result of MDS.
4 MR. WILLIAMS: Off the record.
5 (Discussion off the record.)
6 Q (BY MR. WILLIAMS): You've indicated that there
7 are studies which indicate that there is an increase of
8 MDS at exposure to benzene levels above 100 parts per
9 million?
10 A Yes.
11 Q And you noted, on page 3, two studies which I
12 think are the only two studies in your report that you

did not actually perform, one by Aksoy and one by Kipen?

A Yes.

Q Do you know whether or not there are any other studies which indicate that there is an increased risk of MDS at exposure to benzene levels below 100 parts per million?

A I'm not aware of any.

Q What type of search did you do to determine whether or not that was true?

A I did a search on MDS and benzene exposure and got some classical case reports and so on. There aren't really any. If you go and do a computerized database search, you will not be able to find any study at all on

MDS and benzene exposure.

MR. WILLIAMS: Will you repeat his answer, please.

(Record read by the reporter.)

Q (BY MR. WILLIAMS): Dr. Wong, would you also agree that benzene causes leukopenia?

A Again, within the parameter that we are talking about sufficient exposure.

Q Do you know whether or not Mrs. Lakie has leukopenia?

A I don't remember.

Q Do you agree that benzene also causes, with sufficient exposure, pancytopenia?

A Yes.

Q And it also causes aplastic anemia?

A Yes.

Q Isn't it true that it is now recognized clinically that acute leukemia is often preceded months or years by abnormalities of the blood and bone marrow?

A I'm not a clinician, so I won't be able to tell.

Q You don't have an opinion?

A No.

Q Is it your opinion that one would have to be a

24 medical doctor to be able to respond to that question?

25 A I just don't think you can. I'm qualified to

1 review and critique the literature in that area to be

2 able to answer your question.

3 Q Okay. Let's see if you can answer this:

4 Do you agree that bone marrow suppression,

5 aplastic anemia and myeloid leukemia represents a

6 continuum or spectrum of blood dyscrasias rather than

7 separate or unrelated entities?

8 A Again, I won't be able to answer your question.

9 Q Have there been studies that have shown that

10 chemical workers occupationally exposed to benzene

11 experience significant mortality excess from leukemia as

12 well as the broader category of broad emphatic and

hematopoietic cancer when compared to workers who were not occupationally exposed to benzene?

A I suspect you're quoting part of my paper, written some time ago. In that paper I basically said if you compare the workers who were exposed to benzene to workers who were not exposed to benzene, you do see an increase largely due to the absence of leukemia in the non-exposed group.

Q But your statement went beyond just leukemia to include all emphatic and hematopoietic cancer?

A That's true.

And the increases that I refer to in the broader group -- in the paper, I already said that

increase was driven by the observation of leukemic cases

of exposed group and the lack of leukemia in the non-exposed group.

I just want to be sure you read the entire article and not just the paragraph.

Q Was this article submitted to OSHA or what was the article that you are referring to?

A I'm referring to two articles that I published in 1987. Before the publication of the article, OSHA invited me to testify at 1986 benzene hearing to talk about my study. So I probably had made some statements similar to what I stated in my articles.

Q So when you testified before OSHA, you actually hadn't published your articles yet. And I believe you are referring to two of the articles, in your opinion, aren't you, regarding the industry-wide mortality study of chemical workers occupationally exposed to benzene?

A Yes.

Q Did you actually testify before OSHA or did you just submit a paper?

A I testified in front of what they call an administrative judge.

23 Q Do you have a transcript of that testimony?

24 A I don't have it, but I'm sure OSHA would have one.

25 I just want to make sure the record is

1 clear. Although the publications, the two publications

2 you refer to, have appeared in 1987, the manuscripts,

3 the papers were submitted to the journal long before

4 that. There is a latency between the submitting of a

5 manuscript and the appearance of the paper. I submitted

6 that way back in 1986.

7 Q A year before it was published?

8 A Lucky, if it is one year.

9 Q When were those studies actually done?

10 A Over a period of years starting from the early --

11 either the late '70s or early 1980s.

THE WITNESS: Good time for a break.

(Brief recess taken.)

(Whereupon, Plaintiff's Exhibit No. 5 is marked for identification.)

MR. WILLIAMS: Back on.

Q (BY MR. WILLIAMS): Dr. Wong, on your letter or your report you've already indicated this, that -- why don't you state your opinion for the record with regard to Ms. Lakie's causation of her disease.

A Basically, my opinion is that her exposure was very, very low compared to the studies of the exposures we have in studies of workers with an increased risk of MDS. So based on that consideration, I do not think her MDS was related to her exposure.

Q And you are basing that in part on a comparison to workers, studies of workers who inhale benzene in air; is that correct?

A Yes.

Q Do you know how Ms. Lakie's exposure took place?

A Through injection.

Q Orally, correct?

A Yes.

Q It was not through inhalation, was it?

A No.

Q Do you plan on offering any opinion as to the risk associated with using the Orafix in this case?

A I'm not sure I see the distinction between what I just said and your question.

Q Well, it is your opinion that there is no increased risk of MDS among workers exposed to benzene levels less than 100 parts per million; is that correct?

A Yes, sir.

Q And the basis for your opinion is the studies which you cited in your report, correct?

A The studies that I have done and also two studies that were conducted by others, yes.

23 Q Has a safe level of benzene exposure ever been
24 established?
25 A Safe for what? I guess that's the question

1 because --
2 Q Safe from risk of disease.
3 A If we take leukemia, as an example, some studies
4 have indicated that exposure below 40 ppm years would
5 not increase the risk of leukemia. There are some
6 publications on that. I, myself, have also analyzed
7 from data from NIOSH, the National Institute for
8 Occupational Safety and Health. I analyzed the data
9 collected by them, and my conclusion is if we look at
10 the specific type of leukemia that's associated with
11 benzene exposure, and that is acute myeloid leukemia, it

would take anywhere between 300 to 500 ppm years to have an increased risk.

Q Didn't one of your studies find a cases where a worker was exposed to 0.5 parts per million for 1.2 years?

A When you try to determine what level is safe, you cannot just look at one or two cases.

Q But that's true, isn't it, in one of your studies there was a case of leukemia in a worker who was exposed to 0.5 parts per million for one to two years?

A It is true, but in any industrial cohorts you would have not only chemically related cases, but you would also have the so-called expectant cases, the background cases.

When we say "background cases," those cases are not related to the chemicals in question. And, therefore, you would not be fair to pick one case with the lowest exposure and try to attribute that to chemical exposure. That case would be what we call a background case.

Q Why do you say that that's a background case and not a case of a person who perhaps is more susceptible of the ill effects of benzene exposure?

A When you compare a group of chemical workers with a group of people not exposed to the chemical, benzene in this case, you will see cases in both groups regardless whether that chemical can cause the disease or not. It would be unscientific to simply pick one case and say that case with the lowest exposure is related to benzene exposure.

Q Wouldn't it also be not good scientific practice to say that it is not related?

A You don't know. What you need to do is do what we call an exposure response meta-analysis. In other words, in your cohort, in your group, you divided people

up into several subgroups, smaller groups according to the level of exposure they had, and see when the risk was, when the risk would go up and at what level.

You cannot look at cases. You have to look

at the underlying population at risk. It can be the entire population or it can be subgroups in the population.

Q I want to get back to my original question which started us on this. I asked you whether any safe level of benzene exposure has ever been established.

You would agree that no safe level has ever been established, would you?

MR. WHITNEY: Objection.

THE WITNESS: That's not true at all. I

just said that based on my analysis on the data directed by NIOSH, if we look at AML, acute myeloid leukemia, as the endpoint, you don't have an increased risk number until you reach 300 to 500 ppm years.

Q (BY MR. WILLIAMS): That's for AML?

A That's for AML, yes.

Q Well, doesn't using AML as an endpoint underestimate the risk associated from benzene exposure?

A I don't think by combining those two really makes much sense, because we have a level that's safe as far as leukemia is concerned.

And in my report I talk about MDS. The level would be one hundred ppm.

Q What is your level to be safe for leukemia?

A For leukemia is either 300 or 500 ppm years,

depending on what exposure you are looking at.

Q What is the difference between saying 300 parts per million and 300 parts per million years?

A 300 parts per million by itself is the average concentration of benzene in the air.

Q Okay.

A 300 ppm years is what we call cumulative exposure. In other words, you are exposed to benzene at a certain level over a period. You multiply the concentration by the duration and get ppm years. It is identical to the concept of how many packs of cigarettes you smoke for how long.

Q In your opinion, is there a safe level of benzene in the air for industry?

A Yes.

Q What is that, not using parts per million years but just using an average concentration?

A Again, going back to my analysis on AML, I come to the conclusion that exposure between 300 and 500 ppm years would not increase the risk of AML.

Let's just take the average, 400 ppm years,

as the midpoint. That would be the cumulative exposures. And if we assume someone who works or is exposed at certain levels for 40 years, in order to get the average concentration, we will simply divide 400 ppm

years by 40 years. That would be the total length of somebody's working life.

Q Okay.

A And that would give us 10.

Q 10 ppm?

A I'm sorry?

Q 10 ppm would be the average concentration in air for the worker who is going to work 40 years in that air?

A Did you say 4 or 40?

Q 40.

A Yes.

Q I want to make sure I understand what ppm years is.

A You say 10 ppm multiplied by 40 years gives you 400 ppm years.

Q Now, this is your opinion on safe level. My question really was: Has industry or the government established a safe level of benzene exposure?

A Certainly OSHA has established.

Q And the OSHA is one part per million, is it not?

A One part per million.

Q Is that considered by OSHA to be a safe standard or was that a compromise?

A You would have to depose OSHA.

Q Didn't Peter Enfante and others argue for a level of 0.1 part per million?

A I think we may be mixing a couple of things here. One is what a safe level is based on scientific data, and the other is what is the level that we should use, in policy. Those are two different issues because in policy you want to build in a huge safety factor.

Q Why is that?

A I don't know.

Q You don't know why?

A I don't know why.

Q Well, are you aware of what water standard is established by the EPA for benzene?

A I run it from time to time. I don't know how they arrive at that number.

Q Isn't it five parts per billion?

A I said I don't know what the number is.

Q Wouldn't the broader associated consideration be that the general public is more susceptible, varying levels of susceptibility, as opposed to workers in the

workplace?

A I don't think. As far as leukemia is concerned, I don't think there is any selection criteria that when you apply for a job somehow if you are susceptible to leukemia you get kicked out.

Q But, again, back to this question I'm struggling with, the OSHA level of one part per million in the workplace, that is not accepted to be a safe level for benzene exposure, is it?

A Let's see if this would help you with your question. A lot of times the standards are set based on what we call risk assessment models. And those risk assessment models would give us theoretical risk, not necessary risk that we expect to see.

In fact, if you apply the numbers provided by risk assessment models back to some real studies, you will see that those risk assessment models way overestimate the number of cases you actually observe in those studies.

Q But has OSHA ever said this is the level of benzene exposure, as long as you are below that, there is no risk for anyone getting ill?

A I don't know what OSHA said.

Q Are you aware of any government agency that ever made such a statement or established a standard below which that agency is suggesting that there is no risk from illness from benzene exposure?

A I have never looked into nor tried to understand the rationale behind proposing or setting a standard, a number, because to me when they use risk assessment,

basically, they do not rely on science anymore.

Q So you don't think risk assessment is based on science?

A It is based on mathematical modeling, not based on real data.

Q I understand what you are saying, I think.

Isn't it true that OSHA, to the best of your knowledge, has never said if you have benzene exposure less than a certain level, there will be no risk from disease due to benzene exposure?

A I don't know whether they have said that or not.

Q Do you know whether any government agency has ever said such a thing?

A I don't know.

Q Now, your opinion is that the risk of AML occurs at a threshold between 300 and 500 part per million years and above; is that correct?

A Yes, sir.

Q And your opinion is that at a hundred parts per million concentration in air, and below, there is no

risk of developing MDS?

A Yes.

Q And these are both, I assume, for workers? We are talking about workers now?

A These are based on workers' studies.

Q All right. Well, are you suggesting that if a worker worked for 40 years in a plant where there was a hundred parts per million concentration of benzene in the air, let's say 99 parts per million benzene concentration in the air --

A How much?

Q 99 parts per million.

-- that worker would have no risk of developing MDS, but would have a risk of developing

leukemia.

A I think you give too much confidence to the industrial hygienist. When we say 100 ppm in the air, we really mean in the neighborhood. I don't think we can distinguish 99 ppm from 100 ppm.

Q We'll say 50. Let's get away from the hundred.

Let's say the concentration in the air is 50 parts per million, and we have a worker that works for 40 years.

A Yes.

Q I take it on what you state here today that your opinion is that worker would have an increased risk of developing leukemia because it would be 2,000 parts per million years?

A Specifically AML?

Q AML. But that worker would have no risk of

developing MDS?

A No increased risk of developing MDS as opposed to the risk that you and I have to.

Q The worker would have the increased risk of developing leukemia, AML, but not an increased risk of developing MDS?

A Yes.

Q That's your opinion?

A That's my opinion.

Q Okay. Now, in cohort studies, if there is an absence of association found between an agent and a disease, that doesn't mean that causation for that disease does not exist by exposure to that agent, does it?

A Depends on the study. If the study is large, the people come from different places, and you have exposure data, I think we are -- you are driving at what the criteria for causation are in chronic diseases.

You cannot just simplify the whole situation

by saying if we see an increase or we don't see an increase in the study we stop at that. We don't.

Q So a study alone, if one study alone does not show an association, it is not the end of the inquiry; is that what you are suggesting?

A I would say most likely not, unless the study is

so huge that we can make exception.

Q How huge would the study have to be with regard to MDS, for example, and being caused by or to show an association between benzene and MDS?

A I would say if we end up observing at least ten or fifteen cases of MDS, and we have some good exposure data for everybody in the study, then I would say that study is pretty definitive.

Q Let me give you a hypothetical. If you studied 110,000 workers, okay, and 75,000 of the workers were exposed to benzene and 35,000 were not exposed to benzene, and assume that the study is age-adjusted and sex-adjusted, and also assume that the workers were employed during the same period of time and at the same factories, in doing such a study, if you discovered an increase or if you discovered in the exposed workers that there were several cases of MDS and in the non-exposed workers there were no cases of MDS, would that be a significant finding?

A If they have exposure data, and we can believe in the exposure data, I think that study would add to the literature, but I don't think that study by itself would have sufficient information for me to say yes, I'm going to look at this study only and nothing else.

Q Okay. But the study would indicate, would it not,

that there was an association between benzene exposure and MDS?

A Yes, it would, but I thought the more important question is at what level of benzene exposure. I don't think we are --

Q Well, assuming the exposure data is well chronicled, such a study would be a significant finding, would it not, in the inquiry whether there was an association between benzene and MDS, benzene exposure and MDS?

A I would think so. If the study is done properly, then I think we should look at that, yes.

Q As a corollary, my first question really was, if you have a cohort study and there is an absence, you find an absence of association between an agent and a disease, that study alone does not mean that causation does not exist between that agent and that disease?

A I think I answered that question by saying it depends on how good the study is, how large the study

is, and how diversified the study is in the sense that do you study only one location or do you have different locations.

When we talk about causation there are a number of things that we should look at: How strong an association is, how consistent the result is, and how

specific the result is and so on.

Q Do you know what the actual level established by OSHA is?

A No, I don't recall at this point.

Q Have you read the recent OSHA regulation?

A What's the date of the most recent OSHA?

Q Probably '96.

A '96?

Q I'm sorry. July '95.

A No, I have not.

Q Now, the basis of your opinion is the studies you've cited, all of which you conducted except for the one by Aksoy and Kipen?

A Yes.

Q And your studies were all mortality studies, weren't they?

A Yes.

Q How do you conduct the mortality study? How do you conduct --

A That's the six-million-dollar question.

Q Is it a hard question to answer?

A (No response.)

Q Are there some basic steps to follow?

A Are you referring to a typical mortality study we do in this country as opposed to someplace else? I

mean, narrow it down for me.

Q Well, ones that you did in this case or not in this case, but the ones you referred to in this case.

A The studies that I have done, and I refer to in my report?

Q Yes.

A Okay. Most of them would fit into what we call historical cohort study. What we do is we identify the group of people we want to study through historical employment records by going to a company, go through all their employment records to identify the people exposed to certain chemicals who work there for a minimum of six months or year. That's how we identify the cohort.

Once we have identified the cohort, then we want to find out what happened to them. If it is a mortality study, we would want to find out whether they have died, and, if they died, what they died from.

And there are a number of places we can go

to to find out whether people are still alive or not such as the Social Security Administration, the National Death Index.

Once we find out who has died, then we have to write to each state health department to get a copy of the death certificate.

We get the death certificate, we find out

what the cause of death was and, basically, compile the statistics, compare how many cases of certain cancer that we have in the study, compare that to what we expect to see in the general population.

That's more of the essence of a cohort study.

Q Okay. Do you actually get, you yourself, get the

death certificates?

A Yes.

Q And do you send those to the nosologist?

A I look at them, but the actual code is assigned by the nosologist.

Q The nosologist reviews the death certificates that you procure and assigns a code from the International Classification of Disease; is that correct?

A I think in essence you are correct, but let me just supplement that. Usually, on a death certificate you have more than just one cause of death. You have several causes of death, and the trick is really to be able to identify the underlying cause of death. It may not be the first one on the death certificate or the second one. There is a very compact set of rules that you have to follow to determine which the underlying cause of death is, and, basically, that's the job of a nosologist.

Q So the nosologist has to look at the certificate and whatever is written there and try to ascertain which is the underlying cause of death? Only the underlying cause of death as determined by the nosologist is coded; is that correct?

A In most of my studies, we would code all of the causes of death on the death certificate, and then the nosologist will identify, make a decision as to which one he or she would call the underlying cause of death.

In the analysis, at least in most analyses, we would use the underlying cause of death.

Q Who typically fills out the death certificate of the individuals that you are looking at?

A I guess that would be the physician, the examiner.

Q I understand there is a fair degree of inaccuracy in a lot of death certificates in terms of listing the causes of death; is that a fair statement?

A I would say it is a fair statement for certain

diseases. For cardiovascular disease, sometime the subgroups -- there may be some misclassification based on death certificates. But for most cancers, the diagnosis is pretty accurate based on the death certificate.

Q You have read articles that suggest that 20 to 50 percent of death certificates are inaccurate with regard

to the listing of causes of death.

A But that's an overall statement. That talks about all death. What I just said is that applies --

Q So you agree with that, but you are distinguishing between certain types of diseases that in your opinion are more common in errors such as cardiovascular diseases?

A Right.

Q Any other disease groups that you would suggest as having a wide degree of error in it?

A Probably some ill-defined conditions due to old age. I think at certain points the physician may not be really interested or want to spend the time to find out what the actual causes of death is.

Q Okay. Now, myelodysplastic syndrome is not listed in the International Classification of Disease, is it?

A Different subtypes may be listed under different numbers, different code.

Q But MDS is not considered a cause of death, is it?

A I'm not sure I understand. MDS is not considered to be a cause of death?

Q Correct. That's my question. I'm asking you whether you agree with that. Isn't that true?

A If I understand your question, I don't think it is true because MDS -- certainly, you can put down MDS on

the death certificate and the nosologist, based on that, can assign a code, an ICD code, to that.

Q So your opinion is that myelodysplastic syndrome is a cause of death?

A Yes, certainly can be cause of death.

Q Well --

A I'm missing something. You cannot be asking that question. I must be missing something.

Q Well, MDS is not listed, is not given a number in the International Classification of Disease, is it? I think you already answered that it's not.

A But the subgroups are. For example, refractory anemia, I know it has a specific code in the international classification of diseases.

Q Okay. But MDS does not have a code?

A That, I don't know.

Q You don't know whether it does or does not?

A I don't know whether it does or does not.

Q Do you know what most people that have MDS die from?

A I don't.

Q So you also wouldn't know the various diseases or conditions that people with MDS die from?

A No.

Q Now, after the nosologist codes the underlying

cause of death, are these codes then placed into a computer?

A Eventually they are, yes.

Q And don't you use a computer program to group the various codes of causes of death as they have been put in by the nosologist?

A Sometimes we do and sometimes we don't. Depends on what kind of analysis we are talking about. If we are looking at cardiovascular disease, yes, we are going to group some of those individual ICD codes into broader category. But for cancers, sometimes we look at individual codes.

Q For cancers?

A Yes.

Q Okay. In the studies that you noted in here in your report in this case, didn't you use a computer program by Marsh, primarily, to group these various causes of death?

A Yes.

MR. WILLIAMS: And let me get you to identify this document if you would. This will be Exhibit 6.

(Whereupon, Plaintiff's Exhibit No. 6 is marked for identification.)

THE WITNESS: Is there a question

outstanding?

Q (BY MR. WILLIAMS): Yes. Can you identify the stuff?

A What is that?

Q Exhibit 6, can you identify the document?

A It is a table. Title of the table is "University of Pittsburgh Mortality and Data System, 1995. ICDA Elements of Default Cause of Death List - 63 Causes."

Q Is this the breakdown of causes of death that the Marsh computer program recognizes?

A It is one of the many different systems that he has. If you don't specify it, that would be the default.

Q What does that mean?

A That means when you run your program, you ask if -- you have a number of choices. If you decide not to make a choice, this is the one you end up with.

That's what we mean by "default."

Q But this is one of the Marsh breakdowns of disease, correct?

A It is one of the available type of analyses you can get out, yes.

Q Is this one that you used in your mortality studies?

A No.

Q You did not use this one?

A I did not use this particular one.

Q On this one, is MDS listed as a cause of death?

A No.

Q Is refractory anemia?

A No.

Q If a nosologist saw a death certificate that had MDS listed as a cause of death and nothing else, how would the nosologist know how to code that?

A I don't know. I'm not trained as a nosologist.

MR. WILLIAMS: Let's turn to your studies which will be Exhibit 7.

(Whereupon, Plaintiff's Exhibit No. 7 is marked for identification.)

Q (BY MR. WILLIAMS): The first one I have, "Critical Review of Cancer Epidemiology."

A Is this the one published in the American Journal of Industrial Medicine, 1989?

Q Correct.

Is this one of the studies you rely on for your opinion in this case?

A Yes.

Q I'll state the whole title for the record: "Critical Review of Cancer Epidemiology in Petroleum Industry Employees, With a Quantitative Meta-Analysis by

Cancer Site."

Now, this study looked at the incidence of cancer, did it not?

A Yes.

Q And it wasn't looking specifically at exposure to benzene, was it?

A Only in the sense that refinery workers would be exposed to benzene.

Q What was the research question?

A The research question was to look at a number of cancer sites that have been indicated in the literature to see what sites are elevated.

Q What cancer sites were you looking at in this study?

A Basically, we looked at all.

Q So I take it you were not looking for

17 myelodysplastic syndrome?

18 A No, we were not.

19 Q And you weren't looking for other cytopenias?

20 A No.

21 Q And you were not looking at the increase of MDS as

22 a result of benzene exposure in this study?

23 A No.

24 Q And this study did not also look at women, did it

25 not -- or did it?

1 A Off the top of my head, I cannot tell you whether

2 some of the studies that I include in this analysis have

3 women or not. I have to go back and look at individual

4 studies.

5 Q If you can read this sentence here.

A What page?

Q Page 304.

In the last paragraph, middle of the last paragraph where it says, "The issue of whether benzene" -- I'll just read it, and you tell me what it means -- "or other petroleum hydrocarbons can affect only acute myelogenous leukemia or other cell types as well remains unresolved."

A Yes.

Q Is that contradictory to your earlier statement that in your opinion there has only been an association between benzene and AML?

A I'm glad that you did not depose me six months or whatever earlier because that statement was true at that time because, when we looked at all the data in 1989, we did not have cell-type-specific data. And that's why we make that statement.

Subsequent to that, we looked at that issue, we collected data on cell-type leukemia, and we published our analysis last year.

Q Is this one of the new articles that you brought with you today?

A No. It is one of the articles that I reference in my report.

Q Okay. Would that be the cell-type-specific leukemia analysis?

A Right.

Q This study, you know, you indicated -- this is a meta-analysis where you basically have reviewed other studies?

A Are you talking about the 1989 paper?

Q Yes.

A It is a critical review plus a meta-analysis of all of the refinery studies ever done in this world, including some of them that I have done.

Q What relevance does the study have to your opinion

or to this case?

A To this case?

Q Yes.

A Because we know that it takes more -- it takes a high concentration of benzene to induce MDS and leukemia. And if we don't see an increase of leukemia in the population exposed to certain levels of benzene, then we can safely conclude that we do not expect to see MDS in that group as well. And we can make some

judgment as to people exposed to benzene at those levels where they would have an increase of MDS or not.

Q Okay. Even though these studies were not directed to find instances of MDS, correct?

A That's true.

Q And they were directed to find instances of cancer which Mrs. Lakie does not have; is that true?

A Let me clarify that. This is a 1989 paper, it is a critical review of refinery studies, individual refinery studies. Okay?

Some of those studies, in fact, all of those studies, look at more than just cancer. But the topic of analysis in our 1989 paper was cancer. I just want to make it clear that those studies, the individual studies themselves, did look at more than just cancer. Included in them would be disease of the blood and that would include some of the subtypes of MDS.

Q Okay. But this paper, this '89 paper that you are relying on didn't look at the association of MDS and benzene?

A No.

I missed the last part of your question. Did not look at?

Q Did not look at the association of MDS and benzene?

A That's correct.

Q In fact, it wasn't specifically even looking at benzene, it was looking at cancer sites in refinery workers.

A Right.

Q And on page 304 didn't you also conclude that, in the middle of that same paragraph:

"Third, diagnostic specificity (or lack of) for lymphatic and hematopoietic diseases on death certificates, particularly on older death certificates, may result in inaccuracy of underlying cause of death classification."

A In some of those death certificates in the 1940s,

16 1930s, I would say yes.

17 Q When did most of the people die that you were

18 looking at in these studies?

19 A These studies cover a period from 19 -- I think

20 way back from the 1930s all the way to the 1980s. I

21 won't be able to tell you when most people died.

22 Q Didn't you also conclude in your study that the

23 incidence of cancer was actually less in the population

24 you studied than in the general population?

25 A For certain cancer, yes. For lung cancer, for

1 example.

2 Q Anything else?

3 A There is a small decrease in lung cancer, there is

4 a small decrease in digestive cancer. Those were the

two major sites.

Q So you found that these workers were actually healthier than those in the general population?

A In terms of lung cancer, yes.

Q Is it important when you do a cohort study, and try to apply that result to a case, to have a correspondence of the age group involved in the case you are analyzing?

A I don't think so unless we know that the biological mechanism would be different depending on the age.

Q Well, earlier I asked you what the incidence of MDS in the general population was and you told me that the age group from 60 up was one in a thousand.

Why did you confine yourself to the age group of 60 up?

A I run into that statement someplace. I don't know what that -- I mean, if somebody asked me the question, and I have the time to look it up, I can give you the incidence for every age group. It is just something that I remember.

Q You didn't tell me that because Ms. Lakie is in the 60 age group?

A No.

Q Who engaged you to perform this particular study?

A Who what?

Q Engaged you.

A Engaged me, okay.

Mobil Oil Corporation.

Q How much did they pay you for this?

A I don't remember the number.

Q Do you remember approximately?

A I spent a lot of time on that over a period of three or four months. Beyond that, I really don't remember what the contract figure is.

Q Did you do it by the hour or was it a contract

16 amount?
17 A I think it was by the number of hours I spent and
18 my staff.
19 Q And your staff?
20 A It is not a fixed amount, if that's the question.
21 Q So it was an hourly rate?
22 A Depends on -- it was a time and material kind of
23 contract.
24 Q What did you charge per hour? Was it the same
25 thing that you charged today? I think your rate is 330

1 an hour.
2 A No. That was back in 1989.
3 Q Do you know what your hourly rate was?
4 A I don't know now. Would be less.

Q How much staff did you have working on this?

A On this?

Q How much staff do you have?

A What was your question, now?

Q Back then, what was the size of your staff?

A 1989, probably about 30, 40.

Q And they all work here in San Francisco?

A No. We used to have an office in the East Bay.

Q Alameda?

A East Bay, Alameda.

Q Who would have those figures as to how much you made on this particular study?

A I would not know. Let me tell you why. In 1987, our company, at that time I own a company by the name Environmental Health Associates. Together with a couple other people, we own the company.

We merged our company in 1987 around that time with a much larger environmental engineering consulting firm by the name ENSR, E-N-S-R. And based on the merge agreement, I would work for them for another three years. And I worked for them for three years.

In 19 -- the end of 1990, I left ENSR. And that project was done when I was at ENSR. So I have no idea what happened to all the files.

Q Did they pay ENSR or did they pay you?

A At that time?

Q Mobile Corporation.

A When I did the study?

Q Yes.

A They pay ENSR.

Q Were you an employee of ENSR?

A I was an employee of ENSR at the time.

Q And it also indicates you were executive vice president. Were you a part owner of ENSR?

A It was -- at that time ENSR was a public company.

I own some shares.

Q And they still exist, I take it, ENSR?

A I think so.

Q You know better than I.

A I think so.

Q And on this particular study your opinion of why this is relevant is related to the amount of leukemia you found?

A Well, the reasoning is we all know that it takes more benzene to induce MDS than to induce leukemia. And if I don't see increase of leukemia in this group of

workers, then I know that they will not have an excess of MDS. And knowing something about the exposure level, that would let me say something about what level would

be safe as far as MDS is concerned.

Q And if your hypothesis was shown to be incorrect or if you use a different hypothesis, your hypothesis being that it takes more benzene to induce MDS than leukemia --

A Right.

Q -- if you use a different hypothesis, that it took less benzene to induce MDS than it does to induce leukemia, your opinion would change in regard to this article?

A Oh, yes. If it takes less benzene to induce MDS, and if I'm convinced of that, then the study on leukemia would not give us any useful information regarding MDS.

Q And your sole basis for your hypothesis that it takes more benzene to induce MDS than leukemia is the two articles which you cited by Aksoy and Kipen, correct?

A Those two, plus any articles' summary on benzene toxicity would tell you that it takes more benzene to induce MDS than leukemia.

Q When you say "summary of benzene toxicity," are you referring to a textbook?

A Yes. Or even review documents by maybe even OSHA or EPA.

Q Can you give me an example of an authoritative medical textbook which would indicate what you've suggested?

A I have to go back and take a look at what specifically says that. I mean, I come across that all the time, and I can't give you the name of the book today.

Q When was the last time you recall reading that statement in a medical textbook?

A Long time ago.

Q More than five years ago?

A I won't say that long.

Q You don't know --
A Over the years maybe on and off I would come across that.
Q If I showed you a published peer review article that stated that acute leukemia is often preceded by months or even years of functional abnormalities of the blood and bone marrow, would that cause you to question your hypothesis?
A No.
Q Why?
A Because you may see some cases of leukemia with

MDS, but then there may be some cases of leukemia that does not require the development of MDS.

So what I'm saying is you may see a higher,

high number, larger number of leukemia than MDS.

Q But if the bone marrow disorder preceded the development of leukemia, isn't that contrary to the hypothesis that you have asserted that it requires more benzene exposure to induce MDS than leukemia?

A Yes. But simply, the simple fact that you have abnormal bone marrow doesn't mean that you have MDS.

Q Well, you do agree that MDS was called and still is called a preleukemia syndrome, don't you, in the medical community?

MR. WHITNEY: Objection.

THE WITNESS: I guess that goes back to the previous answer I gave you. Not every leukemia has that occurring before the diagnose of leukemia.

Q (BY MR. WILLIAMS): Okay. But if MDS is known in the medical community as a preleukemia, doesn't that indicate that its development is a precursor in those instances to the development of leukemia?

MR. WHITNEY: Objection.

THE WITNESS: In some of them, not in all of them. Are you saying that every leukemia's got to have

25 MDS?

1 Q (BY MR. WILLIAMS): No, I'm not saying that. I'm
2 trying to obtain an understanding and reconciliation of
3 the description of MDS as preleukemia with your
4 hypothesis that it takes more benzene exposure to induce
5 MDS than it does to induce leukemia.
6 A I guess what I'm saying is that some forms of MDS
7 is. Even if we agree that some forms of MDS is
8 preleukemic, I don't see how that can answer the
9 question, either confirm or refute the observation that
10 the toxologist and epidemiologist have observed that it
11 takes more benzene, higher concentration, to induce MDS
12 than leukemia itself. I don't see a connection.
13 Q Okay. What toxicologists have concluded that?

A In a lot of textbooks and review articles, basically, they list at what level you receive what kind of symptoms. For example, if you are exposed to benzene, tens of thousands ppm, one of the adverse health effect is immediate death. Very, at very low level you can feel lighted-headed. There is a continuum.

And when you look at that, look at those review articles, the level of exposure associated with MDS is much higher than leukemia.

Q But you can't show me a textbook that shows that today?

A I did not anticipate your question. I did not bring a textbook with me to answer your question.

Q Okay. Well, wouldn't you agree that the development of aplastic anemia would require less benzene exposure than the development of leukemia?

A I don't know that.

Q You don't know the answer to that?

A I don't know the answer.

Q And wouldn't you agree that the development of leukopenia would result from less benzene exposure than that which would be required to induce leukemia?

A No. I think more.

Q You think leukopenia, reduction of white blood cells, requires more benzene exposure than development of leukemia?

A I think so.

Q And what articles suggest that?

A Well, again, I can't give you the name of the articles today.

Q Let's go to the industry-wide mortality study. I think parts one and two.

What was the research question in these studies?

A Basically, to look at the relationship between

25 benzene exposure and leukemia. And when we first

1 started the project, we were also interested to get some
2 information on specific leukemia cell types.
3 Q Now, this study didn't include women, did it?
4 A No.
5 Q And it included all young males, did it not?
6 A All young males. I'm not sure. What do you mean
7 by "young"?
8 Q 20s to 30s.
9 A Are you saying that in my study there was no one
10 older than age 40?
11 Q I'm asking you, really,
12 A We have lots of old people.
13 Q In this study?

A Yes.

Q In this study, to be included in the cohort required employment of at least six months, correct?

A Exposed for at least six months.

Q The study -- but you don't know how long these people in the cohort actually worked in the refinery, do you?

A These are not refinery studies.

Q These are chemical workers from seven plants. You don't know how long they worked at the plants?

A Yes, I do.

Q Where is that listed?

A When you say "where is it listed," you mean you expect to see a table of the length of employment of all

7,000 workers?

Q You say --

A I mean, the question can mean anything. I mean, what are you looking for?

Q Well, conceivably some of the people in the cohort could have worked eight months and then left employment?

A We have that information. We would know that.

Q That information is included in one of these tables, I take it?

A Okay. If you go to the second article, page 384, Table 3, that would give you the distribution of the workers by length of exposure.

Q In other words, the majority of workers worked for less than four years, didn't they?

A When you say "majority," I assume you mean more than 50 percent, and if that's the case it is not true.

Q I mean -- okay. The majority worked for less than nine years or less?

A Yes.

Q Is that right?

This study, I take it, does not allow you to

make any statistical conclusions with regard to the effect of benzene on women.

A Why not?

Q There are no women included. It is all men.

A Do we have any evidence that women would behave differently when they are exposed to benzene?

Q Well, are you aware of no differences between the development of MDS in men and women?

A As a result of benzene exposure?

Q In general.

A I thought we are talking about benzene-related MDS?

Q Well, in general, are you aware of any differences between the development of this disease, MDS, between

men and women?

A I'm not aware of any paper that talks about biological differences or mechanisms in terms of developing MDS.

Q Well, this paper doesn't allow you to make any statistical correlations between the effect of benzene exposure on men and women, does it?

A I think it would be a mistake to say that if the study is based on men alone that the result would not apply to women.

Q That's not what I'm asking.

A Can I finish my answer? I haven't finished.

MR. WHITNEY: You can certainly finish

answering the question.

Let him finish his answer. Don't cut him off, please.

THE WITNESS: The very first study on smoking and lung cancer was based on men, and certainly you would agree that simply because the first study was done on men alone that we can't say, "Hey, you are a woman, you can smoke, the result doesn't apply to you."

MR. WILLIAMS: Thank you.

Now, if you can answer my question, I would appreciate it.

Q (BY MR. WILLIAMS): My question was: This study doesn't allow you to make any statistical correlation between the effects of benzene on men and women, does it?

A It does because there is no scientific evidence to say that females react differently than men when it comes to benzene.

Q But this study didn't study women, does it?

A It does not.

Q So based on this study, you cannot make a statistical correlation between the effect on men and women because you didn't study any women?

MR. WHITNEY: Objection. That question has been asked and answered three times now.

Q (BY MR. WILLIAMS): Do you understand the point I'm trying to make?

A I understand the point you are trying to make, but by the same token, we are studying people in New Jersey, we are studying people in Texas. Does that mean that the result does not apply to people in the Bay Area?

Q You can take my deposition at a later time.

A I just want to see the logic of your question.

Q It is really a simple question: You didn't study women?

MR. WHITNEY: I object and I'll instruct him not to answer.

You are arguing with him. He is not going to give you the answer that you want.

MR. WILLIAMS: I don't know what the answer is. Let's try one more time.

Q (BY MR. WILLIAMS): This study did not study women, correct?

MR. WHITNEY: Objection. It's been asked and answered. Go on to your next question.

MR. WILLIAMS: I want an answer to this question.

MR. WHITNEY: He's answered it.

THE WITNESS: I can give him the answer one more time.

I did not study women in this study.

MR. WHITNEY: Let's take a little break.

(Brief recess taken.)

Q (BY MR. WILLIAMS): Now, this study, the one we have been discussing, the industry mortality, I and II, that was studying the benzene exposure in leukemia, correct?

A Lymphatic and lymphopoietic causes of death. Leukemia was the focus.

Q You were looking for the relationship of benzene and MDS, were you, in the study?

A No. Other than we do look at disease of the blood, and some of the subtypes of MDS would be included there.

Q What subtypes would be included there?

A Refractory anemia would be included for sure. I know that's included.

Q And what else?

A I have to look at an ICD book to tell you.

Q Now, the dates of death in the study I believe are from 1946 to 1987; is that your recollection? I'm looking at page --

23 A December 1977, yes.

24 Q Okay. And in the ICD, I think you used number

25 eight.

1 A I forgot whether it was eight or seven. Eight.

2 Q And MDS was not listed as a cause of death in that

3 version of the ICD, was it?

4 A No.

5 Q The computer program you used to group the causes

6 of death was the Marsh version, was it not?

7 A (No audible response.)

8 Q If you look at Footnote 32 on page 380 --

9 A What page?

10 Q 380. I'm looking at Number 1. General result.

11 A Page --

Q 380.

A 380?

Q Yes.

A Yes. I'm looking at that page.

Q Footnote 32.

A Right.

Q Was that the Marsh program you used --

A Yes.

Q -- to code or to group the causes of death?

A Yes.

Q Is that the same program that's been identified as Plaintiff's Exhibit 6?

A Yes and no. It is known by the same name, but the program has been modified over the years. They are

different versions just like WordPerfect 5.0 and 5.1 version.

Q Which version did you use?

A I forgot the version. The most current version at that time. If you ask me what number, I don't know.

Q Isn't the only difference between the version you used, which was 1980, and this one was the addition of AIDS in number 63?

A No, that's not true.

Q What are the other differences?

A If you look at one of the mortality tables, for example, Table 6 on page 371 in part one.

Q Yes.

A And very specifically I would like to point you to one of the causes of death in the table. About midway through, you will see "Diseases of blood." ICD code 280 to 289. Do you see that?

So in that version of the program that I used at that time, it's a specific cause of death called diseases of blood.

Q And --

A In the table that you gave me, that is not there.

23 Q So the version you used corresponds to only the
24 causes of death as listed in this Table 6?
25 A No. It has more causes of death than this, but

1 these are the ones that we present in the paper.
2 Q Okay. And the number in parens next to it, all of
3 these, those were the ICD codes, correct?
4 A In that paper those are the eight ICD codes.
5 Q And for Diseases of blood, 280 to 289, none of
6 them include MDS?
7 A It includes some subtypes.
8 Q But none say myelodysplastic syndrome, do they,
9 280 to 289?
10 A Because that term was not in common use, or
11 accepted use, at that time.

Q Okay. Now, at the top of this table it says "All causes"?

A Yes.

Q Then "Observed Deaths, 1,036." Is that supposed to be a summation of what everything is below it, or is that a separate category?

A The answer to both questions is no, and the reason being all causes of death that would be the number of all the deaths.

Q Okay.

A But as I said, I have not presented all the causes, all different causes of death in the table simply because of space limitation. So if you add up all the numbers, it would not give you that number.

Q Okay. Well, the number I'm looking at is 1,036.
A That would be the total number of deaths that we observed in our study.
Q Well, when I start adding these numbers below it quickly in my head, it exceeds 1,036.
A Oh, okay. If you ask me first, I would save you some trouble. For example, if you looked at "Cancer of digestive system."
Q Yes.
A The 4th line. The ICD code is from 150 to 159, right?
Q Yes.
A And for that group of deaths we have 49 as observed.
Q Yes.
A And under that we have five additional cancer sites within the digestive system and also indented.
Q Okay.
A So that simply means that within the digestive system we also have the number of cancers of the esophagus, number of cancers of the stomach, number of

22 cancers of the liver and so on. And if you add those
23 numbers and also the number for the total digestive
24 system, then you are adding some numbers twice.
25 Q Okay. So the ones that are indented are

1 subcategories?
2 A Exactly.
3 Q Thanks for explaining.
4 "All cancers" was 214 and everything under
5 that up to "Benign neoplasms"?
6 A The best way to look at that is to look at by
7 comparing the ICD code. If the ICD code is already
8 included in one of the ICD codes above, then you assume
9 not to add that one again.
10 Q Now, if a person had MDS and died of another

cause, that would not be reflected in this table, would it?

A If it is the underlying cause of death. Because, for example, leukemia, nobody dies of leukemia as the immediate cause of death. Most of the time people would die from the infection and so on. That would be the immediate cause of death.

But as I said, the nosologist would be able to pick it up.

Q For example, somebody had MDS and developed an infection and died of pneumonia and the death certificate saying underlying cause of death would say pneumonia?

A No. The death certificate would not say the underlying cause of death. That would be the immediate

cause of death.

Q How do you know?

A Because I have seen some death certificates, including on leukemias.

Q But you didn't prepare the death certificates in these cases, physicians did?

A I did not, but I look at them.

Q And some of these people died back in the '40s and '50s, correct?

A Yes.

Q And if the physician was unfamiliar with MDS, which didn't start to be used until the early '80s, the physician might, in fact, have listed pneumonia as the cause of death; isn't that a distinct possibility?

A I think we are mixing up several things here. One is we all agreed that MDS, the term itself MDS was not used, at least not widely used, before the 1980s. So part of your statement is correct.

But the subtypes, the terminology for different types of MDS have been used for a long time. If it is a refractory anemia, then the physician would

22 put down refractory anemia as one of the causes of
23 death.
24 Q But if the cause of death was pneumonia, the
25 physician might also put down cause of death, pneumonia?

1 A Absolutely. It is the job of the nosologist.
2 When you see a combination of different diseases,
3 symptoms, once you have gone through the training you
4 will be able to pick up the pneumonia is the result of
5 the infection as a result of the underlying cause of
6 death. The nosologist's job is to be able to pick up
7 the underlying cause of death.
8 Q And that depends on the accuracy of the death
9 certificate, doesn't it?
10 A Absolutely, yes.

Q And if we take a hypothetical: The death certificate did not say refractory anemia, it just said pneumonia, then the nosologist would list it under here as pneumonia, would he not?

A In your example, yes.

Q Now, in the second part of this article, I believe we already discussed this earlier, this was the article where you concluded that there was a significant increase of risk of leukemia and for lymphatic and hematopoietic cancer as compared to workers with no occupational exposure; isn't is that right?

A Can you refer me to where I said that?

Q Sure. Well, the last page on 395, it says:

"Despite these limitations, the study has shown that chemical

workers occupationally exposed to benzene experienced significant mortality excess from leukemia as well the broader category of all lymphatic and hematopoietic cancer, when compared with chemical workers who were not occupationally exposed to benzene."

A I'm looking for the place where I said that.

Q I'm not sure if your page number is the same as mine. I have the top of page 395, very last page.

A Yes, I have that page. I know where. I found the place where you read that. I'm just looking for --

Q The abstract?

A -- the place where I said that.

That finding was driven by the fact that we saw some leukemia cases in the exposed workers and none at all in the non-exposed workers. And that's one of the major problems in the study. If you rely on a small

21 comparison group, when you observe an increased risk
22 based on a ratio, you don't know whether you are seeing
23 an increase in the exposed group or you are actually
24 seeing a deficit in the unexposed group. I just want to
25 clarify that point.

1 Q And this is substantially the same statement you
2 made to OSHA, was it not?
3 A Yes.
4 Q And these were the studies that you were referring
5 to?
6 A Yes. But by the same time, I also said, remember,
7 whenever there is an increase reported in that study,
8 that increase may be a reflection of the fact that we
9 did not see any leukemia at all in the non-exposed

group.

And, in fact, I have two comparison groups in the study. One, the small group of non-exposed workers and another comparison group is the general population. And when I use the general population as the comparison group, I do not see an increase.

Q You saw an increase with the non-exposed workers, but not when comparing the group to the general population?

A Right. And in the study I said we were hoping for a much larger comparison group than the three thousand or so we ended up with, and we ended up with a much smaller group than we anticipated.

Q Was the group large enough to make any kind of conclusion from, the group you studied?

A The comparison, we are getting confused now. The

risk estimates based on the internal comparison group, the group of non-exposed workers, I don't think that's reliable because the sample is just too small.

Q The sample of non-exposed workers?

A 3,000 is too small.

Q And how many exposed workers did you look at?

A About 4,000.

Q And there were also problems with the problems associated with death certificates, correct?

A In general, yes. I mean, that --

Q That problem being diagnostic accuracy?

A Yes. In death certificates in the '40s and '50s, yes.

Q And there was also a lack of in-depth clinical information as well; is that correct?

A Well, certain questions you want to answer, the study may not be able to answer the question, yes.

Q And this study was funded by the CMA; is that correct?

A The Chemical Manufacturers Association.

Q Did they pay for this?

A Yes.

Q And you were working at that time for Environmental Health Associates?

A Yes.

Q What was your interest in that company?

A That was basically our company. "Our company" meaning the company was owned by the three of us. There were two other principals in that company.

Q Who were the other principals?

A One was by the name Robert Morgan, and the third one is Donald Whorton.

Q Are they scientists?

A One is. I better say they are scientists. One

is.

Q What were --

A One is an occupational physician.

Q Which one is that?

A That is Donald Whorton. Bob Morgan, Robert Morgan has an M.D. degree and also a degree in epidemiology. So he's kind of both.

Q His M.D. and --

A M.D. degree and Master degree in epidemiology.

Q Does the company still exist?

A No. We merged with ENSR, E-N-S-R.

Q And that occurred, I guesses, in 1981; is that right?

A No. It occurred in 1987.

Q Okay. It looks like you started working for ENSR, E-N-S-R, Health Sciences in 1981. Maybe you can clarify

the way that's set out there.

A We started a company around 1981, Environmental Health Associates, and that entity continue until 1987 when we merged with ENSR. Then in 1987 it became part of ENSR.

Q Okay. And do you recall what CMA paid you to do this study?

A I don't remember.

Q Who would have those records? Where would those records be located?

A Maybe ENSR.

Q Okay. So all the financial records related to these studies would still be at ENSR?

A I left everything there.

Q Okay. The Marsh computer program we were referring to, is it possible to retrieve a copy of the one that you used in the study?

A I'm not clear exactly what you want to get.

Q A copy of the Marsh program, hard copy is fine,

that you used in classifying the cause of death in this industry study.

A When you say "the program," what do you mean?

Q You say this is a default program for causes of death unless you choose another one?

A I guess things are a little bit confusing. What

we are looking at, Exhibit 6, is not a program per se. It is simply the grouping of the causes of death that Gary Marsh used in the 1995 version of the program.

If you don't specify which grouping you want, that is not the program. When you say "the program," to me is more than just the grouping of the causes of the death. You would have all the Fortran languages, the software packages.

Q Do you have a copy of the grouping that you used in this industry study?

A I think I may have a copy of the user's manual for the version of the program that I used. And if I still have that, most likely in that manual there would be a table similar to this.

Q Do you think it will show you the various tables or options that one has in using the groups?

A At that time there may not be options.

Q There must be just one grouping?

A Yes. Just one grouping that I used. As we get more sophisticated, it provided more options.

Q Can I get a copy of the user's manual?

A Well, I don't know whether I still have a copy or not. You are talking about 15 years ago, a program that I used. I can check.

Q If you still have a copy of it, can I get a copy

of it?

A I would not make a whole copy of the manual. I would just copy the grouping for you.

Q That's fine. But groupings are listed in the manual?

A Sure.

Q Thanks.

A Now, what I will do is, if I find it, I will make a copy and give that to Mr. Whitney.

Q Exactly.

Proceeding right along here. "Cell-Type Specific Leukemia Analysis"?

A Yes.

Q I believe you referred to this earlier. This is related to the first meta-analysis that we discussed.

A Yes, because one of the questions that we raised was the 1989 analysis that we did, did not really answer the question regarding specific leukemia cell types.

Q So was the purpose of this study to determine

which types of leukemia had an elevated risk from exposure to benzene?

A Yes.

Q And --

A Well, a more limited question than that.

Q Tell me.

A Within the petroleum industry.

Q Within the petroleum industry to determine which types of leukemia were elevated due to exposure to benzene?

A Yes.

Q And this paper does not address the association between MDS and benzene?

A Not directly, but I will give you the same answer,

again, and that is, to me, in my opinion, the level of benzene required to induce MDS is higher than that required to induce leukemia.

Q I understand your hypothesis. But MDS was not what you were looking for when you did this meta-analysis?

A No.

Q As a matter of fact, you were only looking at leukemia, correct?

A Different kinds of leukemia, yes.

Q You weren't looking at any other cytopenias?

A No.

Q And who engaged you to perform this study?

A This was a continuation of the 1989 research. It was funded by Mobil.

Q Okay. And do you recall what they paid you for this?

A I think it is somewhere less than 50,000. I'm pretty sure it is less than 50,000.

Q Was that for time and expenses?

A Time and expenses, yes.

Q And this was paid to your current company, Applied Health Sciences?

A Yes.

Q Are you the sole owner of Applied Health Sciences?

A Yes.

Q I see it is incorporated. Are you incorporated in California?

A Yes.

Q Let's move to another article you cited which was another study called "Health Effects of Gasoline Exposure."

A Okay.

Q What was the research question in this study?

A There were two primary questions. One was does

exposure of gasoline increase the risk of kidney cancer. And the second primary question was at that time, since we are going to spend a lot of time and money studying this question, the kidney cancer question, and since gasoline has about anywhere between two to six or seven percent benzene in it, we might as well look at the question of leukemia as well.

Q So the primary focus was kidney cancer?
A Both. Kidney and leukemia.
Q And it wasn't specifically aimed at benzene exposure?
A Well, for the leukemia question, it was, in the sense that the only chemical substance that we know in gasoline that causes leukemia is benzene.

Q Okay. But there were other qualities in gasoline that might cause kidney cancer; is that your distinction?

A Okay. Let me go back one step.

For kidney cancer, we don't know what cause kidney cancer or what we expect in gasoline cause kidney cancer. The reason we want to study gasoline and kidney cancer was based on some animal data. Animals in the lab exposed to gasoline have some renal cancer. And based on that, we say we better look at whether we see the same effects in humans. So as far as kidney cancer is concerned, we don't have any specific chemicals in gasoline that we suspect.

For leukemia, for example, when I did the gasoline study, it was in the late 1980s. We already have a lot of information on leukemia and benzene, and we know that benzene is the chemical in gasoline that we should be concerned with as far as leukemia is

concerned.

Q This study did not look at the relationship between benzene or gasoline and MDS, did it?

A Not MDS. Again, this is a longitudinal cohort study and you cover a period of 30, 40 years going back all the way to the 1940s. So when you use the term "MDS," I have to say no, because that term was not used.

But, again, some of the subtypes of MDS were included in the category that we discussed before, disease of the blood. ICD code 280 to 289.

Q The dates of death studies were up to what time; do you recall?

A '85. End of '85.

Q So MDS was used by '85, wasn't it?

A Maybe for the last part of 1985. But most of the deaths would have occurred in the earlier -- well, I shouldn't say "most." But the studies covered from 1946 to 1985.

19 Q But the research question, the focus of this study
20 was not on MDS or diseases of the blood, was it?
21 A Disease of the blood, to some extent, yes, because
22 we know that benzene at high enough level can induce
23 different kind of disease of the blood, so we did look
24 at that.
25 Q Now, it didn't look at the relationship of benzene

1 or gasoline and other cytopenias?
2 A No.
3 Q And I take it the tables in here were categorized,
4 causes of death. They were also taken from the Marsh
5 program?
6 A Yes.
7 Q Was that accurate?

Wouldn't that program be similar to this Exhibit 6?

A No.

Q No. Do you know what the data of the category you used in this program was?

A The one that you have in Exhibit 6 is the one that I believe Gary Marsh revised toward the end of 1995, and my study was done before that.

Q Was it only revised to add Aids, those in category Number 63?

A Gary Marsh has four different revisions of ICD code in the table that you have in Exhibit 6, and, basically, he wants to show you the correspondence between different revisions for certain diseases.

Q Your study was published in '93?

A Yes.

Q So I take it you used a Marsh category that was being used in what, 1991? Is that when you submitted

this article?

A That's exactly right. The article was submitted in 1991.

Q So whatever Marsh's breakdown was, that was available in 1990?

A I don't know whether he has a new breakdown in 1990, but whatever the current version we had at that point, we used that.

Q Is it possible to get a copy of that version that you used in this study?

A Again, I will try to go back and see if I have that.

Q Okay. Thanks.

Looking at Table 3 in this study. I don't see diseases of the blood listed on that table.

A I knew you would ask the question, and I have something for you. At the conclusion of this study we

submitted a report, the technical report to the sponsor. In that study the sponsor was the American Petroleum Institute.

In the technical report, we don't have any limitation on space, how long or how much we can include, so we have very detailed analysis. And in that technical report we have disease of the blood, whereas for journal they want to cut down on the size of the

article.

In fact, large journals will have space limitation, and they are saying for some causes of death, if you only have two or three it is not very conclusive anyway, don't report them.

So it is their policy. Either you agree

with that or you don't get it published. So we did not include the full table. We were not able to include the full table in the publication.

But since before coming here today, I suspect we may talk about this, so I make copies of the two tables that you may be interested in.

Q Okay. This is a -- thanks.

MR. WILLIAMS: Let's mark these as an exhibit.

(Whereupon, Plaintiff's Exhibit No. 8 is marked for identification.)

Q (BY MR. WILLIAMS): One of these two pages represents Deposition Exhibit 8.

A The first page in Exhibit 8 would correspond to a more complete version of Table 3 in my 1993 article. And the second page of Exhibit 8 would correspond to a more complete version of Table 11 in the publication.

Q So when you read the published report, you'll see, for example, in Table 11 all the causes of death, 2695.

But when --

A Which table are you looking at?

Q I'm sorry. Table 11.

A Okay.

Q Which will exceed the listings underneath it because you haven't listed all the causes of death in the published article, right?

A Also, there is some overlapping numbers within the table that we discussed earlier.

Q Right. Let me see Exhibit 8.

So I take it the relevance of this study for this case is the inclusion of the category of all diseases of blood and blood forming organs in your initial analysis?

A Right.

Q Again, this was a death certificate study, was it not?

18 A Yes.

19 Q And would be subject to the same limitations that

20 surround the study of death certificates, in other

21 words, diagnostic accuracy?

22 A Yes.

23 Q Let's look at the epidemiological study of

24 petroleum refinery employees.

25 A 1986?

1 Q Yes. Now, this was not a study of benzene, the

2 effects of benzene, was it?

3 A Yes, to the extent that refinery workers are

4 exposed to benzene.

5 Q What is the research question in this case?

6 A To find out whether there is any elevated

mortality in this group of refinery workers.

Q From any cause?

A From any cause of death, yes.

Q So was this to determine whether there was an elevated mortality rate in these refinery workers, but it wasn't directed at finding whether there was an association between benzene and disease in these workers; is that correct?

A Well, certainly the interest is to look at some of the diseases that have been implicated with benzene exposure.

Q Okay. But the specific focus of this study was not to study the effects of benzene exposure, was it?

A I would say yes, because that's the right group to study. If you want to study benzene exposure, where do you find people exposed to benzene? One place would be the refinery.

Q Well, I understand that, but I'm just getting at your research question. Is there an association between

benzene and a particular disease in this study?

A The study would be able to answer at least part of the question. If we see an elevated mortality from certain disease, then we would look at what kind of substances those people were exposed to, looking at their job titles and so on to try to make some sense out of the finding.

Q So then benzene would be a subcategory of that inquiry; is that a fair statement?

A Oh, yes, right.

Q So the main focus of this study was not to look at the effects of benzene on workers, it was to look at the mortality of workers from any cause?

A I would say benzene was one of the major interests in doing the study. Certainly benzene is the chemical that is most obvious to study in a cohort of refinery

17 workers.

18 Q This was a mortality study, wasn't it?

19 A Yes, it is.

20 Q And this study was primarily of men, was it not?

21 A Out of the 14,000 workers in the study, a little

22 bit less than 700 were female.

23 Q Which would be a very small percentage, correct?

24 A Yes.

25 Q And I take it that that percentage would be too

1 small to make a statistically significant statement or

2 conclusion with regard to the effects of benzene on men

3 as opposed to women?

4 A I'm not so sure we need to make the distinction.

5 Q Okay. But if one were to want to try to draw a

correlation between gender and incidence of disease, this study would not allow one to do that?

A No.

Q When did this study end?

A When you said "study end," you mean when I stopped, finished doing the study or when the observation ended?

Q Observation, yes.

A December 1980.

Q And at the end of the study, 78 percent of the people in the cohort were still alive, weren't they?

A Did you say 78?

Q Yes.

A Yes.

Q So that for 78 percent of the cohort, this study will not indicate whether that person may have acquired a benzene-related illness; is that true?

A Up to that point they have not, but we don't know what happened after 1980 in that study.

Q You don't know because all you're studying is the

death certificates, correct?

A I don't think it has to do with either death certificate or medical reports. If you end your observation in 1980, whether it is based on death certificate or based on medical examination, you don't know what happened to that person after 1980.

Q Well, if you did a medical examination of the person in 1980 and you found that they were suffering from pancytopenia or refractory anemia or leukopenia, that would correlate in the tables of December, would it not?

A I guess we misunderstood each other.

Q Okay.

A If we do a study and at the endpoint of the study is some clinical findings, medical examinations rather than death certificates, what I'm saying is, whenever

your observation ends you don't know what happened afterwards. Whether it is death certificate or medical examination, it would be the same.

Q Okay. So in this case you were studying only death certificates,?

A Yes.

Q Isn't that right?

A Yes.

Q So you didn't do any studies in 1980 of the 78

percent of the people who were still alive to determine whether or not they had developed a blood disease?

A No, we did not. But we did update the study since we published the last article.

Q That's the next one?

A That's the next one.

Q But at this point you didn't do that, correct?

A No.

Q And this study was funded by whom?

A Chevron.

Q This is 1986. Do you recall what they paid for this particular study?

A No.

Q Would those records be with ENSR?

A Yes.

Q And I forgot to ask you about this "Health Effects of Gasoline Exposure." That was funded by the API; I believe, you indicated that?

A Yes.

Q Do you recall what they paid your company for that?

A When you say "my company," you may be misleading because the study started in either early or mid 1980s, and when I left, the contract stays with ENSR, but I continue to work on the project.

Q I see that on your footnotes here.

Do you recall what they paid for the study?

A That was a longitudinal study, very decentralized. I think it cost about a couple million dollars.

Q Oh, is that right?

A Yes.

Q How many people worked on that?

A That study took almost ten years to complete, and depending on the stage of the study, sometimes we need to have a lot of people. For example, when we collect the data, basically, we send out a lot of research assistants out to different areas to microfilm the employment records, exposure history and so on. We must have at one time 20 or 30 people working on the project. But toward the end when we do the analysis, we don't

have that many people.

Q So over the ten years it cost a couple million dollars to do the study; they paid a couple million dollars to do the study?

A At least that much, yes.

Q And you left ENSR in '90?

A End of 1990.

Q Do you recall how much they paid your new company, AHS, after you left ENSR?

A A much smaller number compared to the $2,000,000,

because at that time we were down to analysis and writing the report. It was strictly based on how many hours I worked. I would say may be 20-, $30,000 for those couple of years.

THE WITNESS: Off the record.

(Discussion off the record.)

Q (BY MR. WILLIAMS): We already mentioned the updated study.

A Yes.

Q Did that study study the same workers that have been studied in the previous one?

A Yes.

Q And at the conclusion of that study, 71 percent were still alive; is that correct?

A Roughly, yes.

Q Which was about 10,000 people?

A Yes.

Q And this was also a death mortality study?

A The same method as the previous one.

Q Okay. I take it, then, there was no analysis or examination done of the workers who were still alive at the conclusion of the observation period?

A No.

Q And was this study funded by the same group?

A Chevron.

Q Chevron?

A Yes.

Q What did they pay for this updated study?

A Actually, the study, most of the activities were done by the people at Chevron.

Q Most of the activity?

A I should have made it clear. For example, obtaining death certificates, finding out whether people died or not, all those tasks were performed by the in-house people at Chevron. I was involved in analysis, in writing the report.

Q You don't recall what they paid for your involvement?

A Maybe a few thousand dollars.

Q All right. The next study I have in my stack is

called "Long-Term Mortality Study of Oil Refinery Workers. Exposure to the Lubricating-Dewaxing Process."

Now, this study did not include any women, did it?

A No.

Q And this was not a study of benzene as an agent?

A No.

Q So that means it was also not a study of the association between benzene and MDS?

A No.

Q What is the relevance of this study to your opinion?

A This is a subgroup in a much larger refinery study that we have done. Although these people work at the

lubricating-dewaxing units, the fact that they work in the refinery, the fact that they might also have worked at other units before they come to the lubricating department, they might have some exposure in the past to benzene.

Q But this paper does not examine what that exposure is, does it?

A Not specifically.

Q So you have no idea of knowing what their exposure was or was not to benzene?

A No. Well, we can read the statement that they were exposed to benzene to some extent.

Q But you don't know how much or how long or when?

A That's correct.

Q Who commissioned this study?

A Gulf Oil Company, but now it became part of Chevron.

Q At the time of the study it was Gulf?

A Correct.

Q And that was when you were with EHA?

A Yes.

Q Do you recall what they paid for it?

A No.

Q Those records would be with ENSR?

A I would think so. I don't know for sure.

Q But you don't have them?

A I don't.

Q You don't have records of what they paid for the studies?

A No.

Q Next study I have is "Retrospective Mortality" study.

Before we get to that, there is a letter to the editor which you also submitted. This was not a peer review submission, was it?

A Most letters to editors are also subject to review by the editors or maybe by one or two reviews.

Q Do you know whether this one was?

A I'm pretty sure it was because we sent a manuscript and it came back and we had to make some changes.

Q That process would be different than peer review of an article for publication, wouldn't it?

A I'm not sure there would be any major difference. A peer review article is usually something that is based on your study. A letter to the editor is something that

you want to talk about regarding either somebody else's study or you want to supplement somebody's paper. And another major difference is the length of the article.

Q This letter apparently deals with lung cancer, doesn't it?

A No. If you look at Table 2 we have quite a few causes of death.

Q None of the causes of death include MDS or blood disorder, does it?

A That was not the focus of this paper. In fact, if you look at Table 2, there is one column called "Richmond Refinery" and the numbers in that column basically are the same as those in 19 -- the paper published in 1986.

Q You mean it is a repeat of those numbers?

A Yes.

Q Which paper are you talking about?

A The Chevron study, the title of that article is "Epidemiological Study of Petroleum Refinery Employees," published in 1986.

Q Okay. The next article is the "Retrospective Mortality and Medical Surveillance Studies of Workers in Benzene Areas of Refineries."

This was a study of all male workers, correct?

A The reason it took me so long to answer your question, there are two parts in the study.

Q My question was directed to the first part, but go ahead.

A The mortality?

Q Yes.

A The mortality part, you are right. They are all male workers.

Q There were women in the second half of this?

A I'm not sure. The second half was on morbidity, on blood cell counts and so on. Those were based on medical records since the 1970s and '80s. So by that time we should have some female workers at the refinery. So there may be a small percentage. I'm not sure.

15 Q The actual article does not say; is that right?
16 A I tried to go through that quickly and see if I
17 can answer you. I cannot at this point.
18 Q Okay. Referring to the mortality aspect of this,
19 the causes of death identified in the article do not
20 include diseases of the blood, MDS or other cytopenias,
21 do they?
22 A No.
23 Q There are only 34 deaths total, right?
24 A It was a very small group.
25 Q Isn't that too small to draw any conclusions from,

1 34 deaths?
2 A It is a very small study because we are focusing
3 on the people who work specifically at the benzene unit.

That's all the people we have from that refinery.

And in answer to your question, yes, it is very small.

Q And the smaller a study, the less power the study has; is that a fair statement?

A Yes.

Q Now, in the second half of this, the medical surveillance program, I believe 303 workers were observed?

A (No audible response.)

Q Is that right?

A It was a question?

Q Yes. You observed 303 workers, correct?

A Where do you get that number?

Q I got that from page 690, in the right-hand column, halfway down.

A On the left-hand column?

Q No. Right-hand column. Sorry.

A 303, yes. I found that number.

Q Okay. And what was the research objective of this aspect of the program?

25 A This paper was more focused on the issue of

1 benzene. But the first part of the study, the mortality
2 study of a small group of people who work at the benzene
3 unit, as you pointed out, that part was just too small
4 to make any definitive statement.
5 But this study also tells us all the blood
6 chemistry results of refinery workers over a period of
7 at least two or three decades. So that's the part that,
8 that's the kind of data we don't have in most mortality
9 studies. In this study we do have blood study chemistry
10 results.
11 Q Does the study indicate how long the 303 workers
12 worked at the plant?
13 A No, it did not.

Q And it also doesn't indicate how many times each worker was tested, does it?

A We should have an average because we know how many individual results we have. We have multiple testings on the workers.

Q On all the workers?

A Yes.

Q Well, the total number of examinations was 1404, correct?

A Yes, about 1400.

Q So if you had 300 workers, that would be a little over four, that would be less than five tests per

worker, correct?

A Yes.

Q And this study says that the blood tests were performed on each benzene worker up to four times a year?

A Up to, not everybody test for four times a year. Up to four times, but not everyone was tested four times.

Q So with regard to the medical testing, this article doesn't indicate how long workers were at the plant or how many times they were actually tested; isn't that correct?

A The report does not tell you that on an individual basis. But we know the total number of tests, we know how many were included in the sample, so we know the average.

Q The average would be four tests?

A About four, yes.

Q And if all the workers were tested four times the first year, that would comprise all the tests done?

A That's correct, but not everybody was tested four times.

Q But this doesn't show when they were tested or how long between tests they were or any other types of data,

25 does it?

1 A Not to that detail.
2 Q Doesn't that present a problem in reaching any
3 conclusions in regards to this test?
4 A No. Basically, what we want to find out is do we
5 have any findings that indicate that we have a major
6 problem. Do we have a lot of low counts.
7 Q Wouldn't you agree that the sample is too small to
8 make any definitive statements about causation between
9 benzene and MDS?
10 A No. We are talking about 1400 measurements.
11 Q Sample people, 300 people?
12 A Yes, 300 people.
13 Q Isn't that sample too small to make any definitive

14 statements with regard to causation between benzene and
15 not MDS, but any cytopenias?
16 A No, not at all because we have 1400 blood
17 chemistry measurements, and if those measurements were
18 depressed, we should be able to see that.
19 Q But you have 1400 measurements that you don't know
20 when they were done or who they were done on; isn't that
21 true?
22 A It doesn't matter. If we see a lot of the results
23 were depressed, if there is a major problem, we would
24 expect to see that. And we can identify which group,
25 where the depression come from and so on, but in this

1 case we don't see that.
2 Q We just found one multiple myeloma in this group?

A So, we find multiple myelomas in lawyers and epidemiologists.

Q How many cases of depressed red blood cells would you expect to find among 300 people to consider that to be a significant finding?

A Well, you look at the average. If we have any major depression, the white blood cell count would be low compared to what we know. And in this case it is about 7,000. I'm looking at page 10.

Q Table 2, right?

A Table 10, yes.

Q So you are looking at the average of 1400 tests for your conclusion; is that right?

A Yes.

Q So you have no way of knowing whether someone was tested once or never tested again?

A True.

Q Well, if someone was tested once and left four years later, and not having been tested again, and in fact had reduced, had anemia, microcytic anemia, that wouldn't show up in your test, would it?

24 A That's true.

25 Q I think that's the end of your articles. I think

1 that's all of the articles you cited; is that correct?

2 Do you have any articles of your own studies

3 that you relied upon?

4 A The Kipen article we talked about.

5 Q No. Any of your articles?

6 A Oh, any of mine. Are you implying that I haven't

7 done that much?

8 Q Too much. I had to read stuff on the plane this

9 morning.

10 A That's all the papers that I have cited, that I

11 have done.

12 Q Let's turn to the next paper which is the Aksoy

paper. This paper is dated 1972, correct?

A Yes.

Q This is well before the nomenclature has changed with regard to MDS; is that right?

A Yes.

Q Now, reading your statement on page 3 of your report, you state that you cite Aksoy as an example, and you say that in this study Aksoy reported:

> "blood-related disorders including decreased WBC in Turkish shoemakers who were exposed to air benzene levels as high as 150 to 650 parts per million."

Is that right?

A Yes.

Q Does the Aksoy article state that exposure to levels less than one hundred would not result in blood-related disorders?

A The cases that he observed all have exposures in that range.

Q Yes. So the exposures he was studying were between 150 and 650?

A Yes, according to his paper.

Q So did he conclude that if you were exposed to less than 150 ppm that would not result in blood-related disorders?

A I don't think he has any case with exposure less than that.

Q But did he conclude in this study, this study is on people who were exposed to the levels that he found which was 150 to 650; isn't that right?

A Right.

Q He doesn't conclude that if you had exposure at less than this level that you would not acquire a blood-related disorder, does he?

A That's true.

24 Q But you concluded that from this article?

25 A Well, the statement I make regarding the Aksoy

1 study was that Aksoy reported blood-related disorders,

2 including decreased white blood cell count, among people

3 who were exposed to benzene levels as high as 150 to 650

4 ppm. That's what I said.

5 Q But you agree that this study does not mean that

6 exposure, this study by itself, does not stand for the

7 proposition that exposures of less than that, you won't

8 develop blood-related disorders?

9 A This study alone would not tell us exposure below

10 the range that he didn't observe.

11 Q Aksoy had some other studies on leukemia and

12 cytopenias and shoe workers, doesn't he?

A Aksoy has written many, many papers based on different combinations, permutations of the cases that he saw in his clinic. So if you are asking different publications, I would say yes, he has written most likely more publications than the number of cases he has. But are they all different studies? I don't think so.

Q Well, have you read the other studies?

A I have read some of his other papers.

Q Did you read the "Follow-up study on the mortality and the development of leukemia in 44 pancytopenic patients with chronic exposure to benzene"?

A If you can let me see, maybe it will help me.

I think I have seen this paper before.

Q Do you recall reading that?

A I think so.

Q Why didn't you cite this particular paper?

A No particular reason. I mean there is some measurements, he makes reference to some benzene exposure in the one that I cite.

Q Okay.

A And the one I cite would be more closer to the case that we have because we are talking about -- well, he talked about blood changes in the 1972 paper as opposed to mortality in the 1978 paper.

MR. WHITNEY: Actually, would you mind making that an exhibit, so we have a complete record?

MR. WILLIAMS: You have the paper. I think it is in Callendar's.

MR. WHITNEY: Okay.

MR. WILLIAMS: If that's what you are wondering. I'm sure it is in Callendar's.

MR. WHITNEY: Okay.

Q (BY MR. WILLIAMS): Do you recall reading the other publication he had in 1974, "Leukemia and shoe

workers exposed chronically to benzene."

Let me show you this also.

A Yes. I've read this as well.

Q Did you read that recently or in preparation for this case?

A Probably not in preparation of this case because the paper, the focus of this paper is really leukemia.

Q Well, doesn't it say at the top "26 patients with acute leukemia or preleukemia"?

A Yes. It doesn't say MDS either.

Q Well, as we already established, MDS has been called preleukemia, hasn't it?

A No, we have not established that.

Q You didn't agree with that statement?

A Only certain types. Not all MDS. I don't think I ever said today that I agreed to the statement that every type of MDS.

Q Isn't MDS, 5Q minus, hasn't it been referred to as a preleukemia syndrome in the medical literature?

A I mean, are you including everything under the sun? I don't know what that means. All I can tell you, that is specifically for 5Q minus. The type of percent of patients who actually get acute leukemia is very low compared to other subtypes of MDS.

Q But are you saying you don't know what my statement meant, my question? My question was hasn't MDS 5Q minus been referred to as preleukemia in the medical profession?

MR. WHITNEY: Objection. He answered this question many times.

MR. WILLIAMS: He said he doesn't know what I mean.

MR. WHITNEY: He answered the question many times already.

THE WITNESS: I'm not aware of any statement or publication that refer 5Q minus as preleukemic, and even if that was the case, I would disagree.

Q (BY MR. WILLIAMS): But you don't have training as a medical doctor, do you?

A No, I don't. But on the other hand, if you look at 5Q minus patients, you don't see a majority of them transforming into leukemia. That means it is not.

Q What did the majority of them die from?

A I don't know.

Q Okay. Let's turn to the next article that you cited which is the Kipen article.

Now, why did you cite this article?

A Well, you are provided with, for one thing, measurements of white blood cell count in connection with benzene exposure.

23 Q And were the authors able to reach any definitive
24 conclusion in this study?
25 A Okay. What they are saying is they see a very

1 good relationship between benzene exposure and, for
2 example, white blood counts before 1948.
3 Q Do you take that to mean reduction in white blood
4 counts?
5 A Yes. The benzene exposure goes up, the blood
6 count, the blood count goes down, yes.
7 Q Wouldn't that indicate the opposite from what you
8 cited in your report?
9 A Absolutely not.
10 Q Why not?
11 A Because if you look at Table 2 --

Q Wait a second. Figure 2 with the graphs?

A Table 2.

Q Okay.

A For workers exposed to air benzene levels above 100 ppm, white blood counts would be slightly reduced at about 7,000. And if you look at that, the numbers in Table 2, you see that there may be a slight depression for the first two or three years. And the exposure associated with that is around 100 ppm.

So that study actually supports the statement that --

Q I'm not looking at the same table as you. Let me walk over there and see.

A Either we are looking at different articles or you

are too tired or I'm too tired.

Q Or all three.

A Table 2. Table 2.

Q Okay.

A Table 2. You look under the column of white blood count. There are three years where the white blood cell count were below 7,000.

Q Okay. I see that.

A And if you go across you will to look at the exposure involved in those years, it will be above 100 ppm year.

Q But the exposure in Approach A is much less than that, isn't it?

A Well, the study also concluded that Approach A is not valid.

Q Don't they actually conclude that this analysis does not conclusively establish that the Approach B estimates are superior to the A estimates? If you look at page 204.

A Where on page 204?

Q Third paragraph from the right side.

22 A Okay. Well, the very last sentence of that
23 paragraph says that Approach A estimates seem
24 exceedingly low during the 1940s. And the generally
25 higher Approach B estimates are likely to be more

1 accurate during those years.
2 Q You would agree that the authors are unsure of the
3 accuracy of either one of these exposure estimates,
4 would you?
5 A They are more sure about B than A.
6 Q But they are actually unsure about both?
7 A It is not black-and-white type of answer. When
8 you talk about historical exposure, nothing is 100
9 percent accurate. That's why when you ask me the
10 question this morning or earlier this afternoon, instead

of using 100 ppm that I used in my report, I remember you used the number 99. It is not accurate to that extent.

Q There were also some problems in the study with collecting the blood data, weren't there, if you look on page 205?

A Okay. Will you direct me?

Q On the top of the first full paragraph on the left side.

A Yes. Specifically, it talk about some of the problems in using hemoglobin or red blood cell count and that's why in my report I refer to the white blood cell count.

Q Don't they also indicate that there was some potential surveillance problems of the workers as well

as estimates of exposure after 1946?

A Well, I'm sure in any study you have some problems in the data, but by and large this is a larger scale study as opposed to some case report that we have seen.

Q Wasn't the results of this evaluation used in part to support the one-part-per-million OSHA standard?

A I don't know. This was published in 1989, and are you referring back to the hearing in 1986?

Q Well, didn't Kipen and Goldstein have an earlier report in this same study?

A No. If that's the case, most likely they will cite that in their paper. Biggest one reference, Number 7, it may be based on the same data. I'm not sure.

Q Okay. So you don't know whether the valuation in this study was used in part to help set the OSHA standard of one part per million?

A No, I don't.

Q I want to ask you about the last sentence in the report and ask you what you think it means.

"We are unable to say that

22 individuals will not experience
23 significant blood dyscrasia at
24 these lower benzene exposures, but
25 based on this data overall

1 population averages cannot be
2 relied upon to detect reasonable
3 deviation from currently permitted
4 exposures."
5 A Remember, "lower" is relative. Here, what we are
6 talking about, people exposed to -- if you go back to
7 Table 2, again, the level of exposure we are talking
8 about range from 32 ppm all the way to 37 ppm.
9 I think we are referring to after 1941 or
10 '42. When the level drops below 100, they don't see any

depression of while blood cell counts. In that context "lower" means anywhere between 32 up to 100 ppm.

Q So you think that's what they are referring to when they say "lower"? They are referring to the lower end in that scale in the Approach B?

A The lower range of exposure in their study. I can tell you for sure that by "lower" the authors in this article do not mean the kind of exposure we see in this case.

Q You think they are referring to the amounts in the table? I understand what you are saying. They are referring to the amounts found in Table 2 or identified in Table 2?

A Yes.

Q And what they are saying, aren't they, is that

they, the authors Goldstein and Kipen, are unable to say that individual will not experience significant blood dyscrasias at these lower benzene exposures. Isn't that what they are saying?

A I can see why you are having a problem, but when you look at the data, clearly it shows that after 1942 when the exposure level was below 100, you don't see any white blood cell depression.

But I have to agree with you, I don't know what the "lower" means.

Q So you are unsure also with the last?

A I'm not sure what that statement refers to. I thought when you say "lower" in your own study that would refer to the lower range of the exposure that you observe. But if that's the case, the data that they have would contradict that statement.

Q Do you agree that some people are more susceptible than others to the effects of benzene toxicity?

A I don't know.

Q You don't know whether they are or not?

A I haven't seen any scientific evidence to support that.

Q You are unfamiliar with any studies which discuss the variance in susceptibility of individuals to benzene toxicity?

A No, I am not.

Q Assuming that some individuals are more susceptible to benzene toxicity, wouldn't that have an effect on your opinion in this case?

A I would say no.

Q Your opinion is based on studies of occupational industry workers, correct?

A Yes.

Q And it is not based on studies of the general

population?

A But there's no selection criteria of individuals in the general population into the working population based upon so-called sensitivity to benzene. It doesn't make sense, because when you apply for a job to work in the petrochemical industry, they don't test you for sensitivity to benzene. If you have a cardiovascular condition you may not get a job. I can understand that.

Q So you have no opinion on whether some people are more susceptible than others to the effects of benzene toxicity?

A I haven't seen any scientific evidence to offer that.

Q Do you plan to offer any opinion at trial on the susceptibility on individuals?

A Just as I said, I have not seen any scientific

evidence to demonstrate that's the case.

Q In preparing for this case, have you looked for that particular point in the literature?

A Not in particular with this case, but in general, the healthy selection, in fact, the selection problem, I'm always interested in that.

Q Could you do a study, an epidemiology study where you took a group of people and put Orafix Special with benzene in it in their mouths and study that group, and then another group of workers or individuals that you did not apply this substance to and compare the effects of that exposure between those two groups?

A Can I do a study like that?

Q Could one?

A Theoretically, yes.

Q Practically?

A Basically, you are talking about almost a clinical trial.

Q I guess that's true.

A So you can design such a study.

Q Would it be profitable for a scientist to design a study?

A Depends on what question you want to ask. I mean, when you do a study, you have a question in mind and whether we want to do such a study or not, whether it is

appropriate or not, depends on the question.

Q Well, the question is is there any adverse effect from benzene in a product that's placed in people's mouths?

MR. WHITNEY: Objection.

Q (BY MR. WILLIAMS): Use that as a question. Would it be proper for a scientist to conduct such a study placing benzene in peoples' mouths to study the effects?

A If somebody asked me that question, what I would

do is to analyze the range of exposure you would get from using Orafix.

Q But you wouldn't actually put benzene in someone's mouth to do such a study; is that what you are saying?

A When you say "put benzene into somebody's mouth," it sounds horrible.

Q Why?

A But in reality, whenever you put something foreign into somebody's mouth, that doesn't sound kosher to me. But we ingest benzene all the time. I don't know what breakfast you have, if you have scrambled eggs you have benzene. There is a substantial amount of benzene in scrambled eggs, in fish and everything. It is a natural occurring chemical.

Q How much benzene is in eggs?

A I don't know. The number is actually pretty high.

In fact, I may know.

In eggs, this is according to U.S. EPA, cited by the International Agency for Research on Cancer, in eggs, 500 to 1900 micrograms per kilogram.

Do you drink Jamaican rum? 120 micrograms per kilogram. Beef, 19 microgram per kilogram.

Q Just so I have a point of reference. 500 micrograms per kilogram, how many parts per billion would that be?

A Kilo, that's 10 to -- it is 6 o'clock. The numbers are getting fuzzy.

Anyway, I'm not going to put myself on risk doing some numerical calculations at 6 o'clock.

Q Would you agree that humans should avoid exposure to benzene?

A If it does not have any health consequence, I don't think -- number one, I don't think you can avoid benzene if you want to live in this world.

I don't see what that question will get you.

Q To the extent possible, should a person avoid exposure to benzene? For example, would you avoid washing tools with gasoline in a closed room for long periods of time?

A No, because the exposure resulting from that kind of activity would be high.

Q It would be a high exposure and you would want to avoid that?

A I would like to avoid that. But if you say do you want to avoid benzene by not eating any more eggs or drinking any more rum, my answer is no. I'm going to eat scrambled eggs and drink rum.

Q My question is, to the extent possible, should humans attempt to avoid exposure to benzene?

I'm not talking about eating food that people eat all the time.

A It is possible for me not to eat scrambled eggs. I guess I don't follow your question.

Q Well, the exposure is so low you are not worried about the risk?

A Exactly. I think you get it.

Q But the same is not true for using gasoline to wash tools in a room. Exposure is high?

A Right.

Q So don't you think it is wise to avoid those types of exposures?

A Absolutely.

Q You work as a consultant for several of the oil companies; is that accurate?

A Yes.

Q Who do you work as a consultant for?

A Who?

Q Which companies, which petroleum companies?

A Over the years or now or --

Q Over the years.

A Over the years, Mobil, Chevron, Gulf before it became Chevron, part of Chevron. Texeco, Amoco, Shell.

Q Do you work -- I'm sorry.

A Most likely I worked for Exxon, too. Basically, I have done a lot of studies in the petroleum industry.

Q Have all the studies you done in the petroleum industry been at the behest of either the API or Chemical Manufacturers Association or petroleum companies?

A Yes.

Q How many studies do you think you have done in the petroleum industry?

A How many studies I have done in the petroleum industry?

Q Yes.

A I don't remember. Twenty, thirty. I don't remember.

Q Do you currently serve as a consultant for any of these companies?

A When you say "currently," it is difficult to answer the question because --

Q I would say in the last --

A Go ahead.

Q In the last four years?

A In the last four years, for sure not Gulf because it is no longer there. I would say Chevron, Mobil, Amoco. At least those three.

Q Do they pay you on a per-job basis, or do they pay you a retainer? What's your relationship with those

companies?

A It is based on time and materials.

Q So they may call you to ask you to do something, and you bill them on time and materials; is that right?

A Correct.

Q I'm looking at the case that you indicated that you have testified in.

A Yes.

Q There were two in 1995?

A Can I take a look? I don't have them in front of me.

Q Exhibit 2.

A Okay, I have it. Yes.

Q Okay. Were those the only two cases in 1995 or were there others?

A Those were the ones that I could remember when I compiled this list. I don't have a place to go to.

Whatever I remember at that time when I prepared the reports.

Q So when you prepared the report, you just thought off the top of your head?

A As many as I could remember.

Q You don't have a record of the cases you testified in?

A No.

Q And I notice the Lakie case isn't on here.

A The Lakie case? I'm sorry.

Q Yes.

A At that time I don't think I had testified in the Lakie case, have I? Today's the first time I've testified in the Lakie case.

Q I notice another case here.

A I'm getting confused. I thought I understand the term "testify" meaning in deposition or trial.

MR. WHITNEY: You got it right.

THE WITNESS: Because he is the lawyer, I'm not.

Q (BY MR. WILLIAMS): in '94, I see on here, you have Girdry versus SmithKline Beecham.

A Yes.

Q I believe you testified in that case, at least in deposition?

A They took a deposition, yes.

Q Do you recall what you submitted to SmithKline Beecham on that case?

A No.

Q Do you have records that indicate that?

A 1994, no.

Q You wouldn't have records that would indicate it?

A No.

Q What happened to them?

A They pay all their bills.

Q And you threw everything away?

A Yes.

Q So you don't keep records of what you did or the bill you sent in 1994 for SmithKline Beecham?

A No.

Q How about for Cavanaugh versus SmithKline Beecham?

A That was also last year's. That was last year.

Q '95?

A '95, yes. And I think a deposition was taken after I had prepared this report.

Q Okay. Do you recall what you billed SmithKline Beecham in that case?

A I did not work on that case that long because it was a very short -- I don't remember how much. But if it is 1995, we may still have the invoices.

Q Can I get a copy of what you billed in that case?

A I'll go back and find them, yes.

Q How much of your time is spent in litigation matters?

A On the average I would say about 25 to 30 percent.

Q Has that changed over the last five years?

A It fluctuates. Sometimes it is less and sometimes it is a little bit more.

Q So your estimate is about 25 percent?

A 25 to 30.

Q Are these probably for defense, defendants?

A Yes. Once in a while I testify for plaintiffs, too.

Q Would you estimate what, 90 percent for defendants?

A I would say so, yes.

Q I take it you have an accountant that does your tax returns?

19 A Yes.

20 Q How do you report the amount you made from

21 litigation matters to him? Is it broken down into

22 categories?

23 A No.

24 Q You just send him bills that have been paid and he

25 figures out your tax returns?

1 A I'm not sure I understand your question.

2 Q You have an accountant that does your tax returns,

3 correct?

4 A Yes.

5 Q For you and your company?

6 A Yes.

7 Q How is income that you received from litigation

matters sent to your accountant? How is it provided to your accountant?

A We report the total number of -- total income for a specific period of time.

Q So it is not broken down?

A No, it is not.

Q Have you read, do you plan on offering any opinions in this case on rebuttal to any of plaintiff's experts?

A I'm not sure exactly what that means. Depending on what questions they ask me. I'm looking to Mr. Whitney for guidance. I don't know what else he would ask me to do.

Q Have you been asked to render any opinions in rebuttal to any of plaintiff's experts?

MR. WHITNEY: In other words, we have the right to have you speak regarding the basis of opinions of the various causation experts of plaintiff.

Q (BY MR. WILLIAMS): Up until today have you been asked to do that?

A No.

Q Have you reviewed any of plaintiff's experts' reports in this case?

A Yes.

Q Do you know which ones you reviewed?

A I don't remember their names. I did that several months ago.

Q I'll assume it is in here, the ones you reviewed.

A I see two names here. Dr. Blanke and Dr. Saady.

Q What did you review from them?

A Deposition transcript.

Q Anything else?

A Dr. Jacobus' notes -- oh, that's our guy. Okay. Okay. Blanke's report.

Q What date is that?

A July 20th, 1995. Dr. Callendar's, August 15th, 1995. And Dr. Swerdlow, S-W-E-R-D-L-O-W. And I'm not sure whether there is a date here. The report is not dated, but the fax says August 17, '95.

I think that's it.

Q Okay. Do you have any opinions today with regard to the materials you reviewed from those experts, other than what you stated in your deposition thus far?

A Other than, obviously, we have different opinions overall.

Q Anything additional to what you already stated today?

A I have not done any detail critique of their report, if that's what you are asking.

Q I want to know if you have any additional opinions with regards to the reports other than what you stated in your report and what you testified to today at this time.

A I don't recall. Not at this point because I reviewed those documents almost six months ago.

Q You haven't seen them since?

A I have not read them again.

Q Other than the articles which you referenced, are there any other articles that you are relying on for your opinion that you stated today?

A Those are the major ones. Of course, there are articles on the general issue of epidemiology or on benzene and leukemia and so on. I'm not so sure I can answer your question with a yes or no.

Q Any particular ones that you haven't discussed today that you plan on relying on?

A No. Unless the question requires me to go to some other documents, rely on some other documents.

Q You had one other document, I believe, that -- I just want to take a quick look at. The IARC document?

A That's the document that you should really have. It tells you how much benzene you have when you eat your scrambled egg.

Q You didn't list it in your report.

A To be quite honest with you, I think last week Mr. Whitney called me and said do I know of any data, handy data that talks about benzene exposure, non-occupational exposure. And that's a very convenient place to go to.

Q And this is, this document entitled "IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans," and there is a chapter on benzene, is what -- that was your response to that inquiry?

A Right.

Q Okay. I'm just going to leave that with your

other articles and have the whole thing on an exhibit.

MR. WILLIAMS: Let's just go off the record for a minute.

(Discussion off the record.)

Q (BY MR. WILLIAMS): One other clarification. Do you mind checking to see what Marsh printouts you have of the various disease classifications that you used in all of the reports that you cited? I'm not sure if I

asked you on each one whether you could provide that or not.

A I will go back and look through and see how many different versions I have that use this menu, and whatever copies I have, I will make copies of the disease groups for you.

Q Okay. Just the ones you used in your reports.

A I will.

Q That will be great.

MR. WILLIAMS: No more questions.

MR. WHITNEY: Mr. Wong will want to read his deposition transcript and sign it.

(Whereupon, the deposition of OTTO WONG, Sc.D., F.A.C.E. was concluded at 6:30 p.m. this date.)

--- oOo ---

I certify under penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Date

OTTO WONG, Sc.D., F.A.C.E.

--- oOo ---

STATE OF CALIFORNIA)
) ss.
COUNTY OF SANTA CLARA)

I, BARBARA FRIEDMAN, a Certified Shorthand Reporter in and for the State of California, hereby certify that the witness in the foregoing deposition,

OTTO WONG, Sc.D., F.A.C.E.,

was by me duly sworn to tell the truth, the whole truth and nothing but the truth in the within-entitled cause, and that the foregoing is a full, true and correct transcript of the proceedings had at the taking of said deposition, reported to the best of my ability and transcribed under my direction.

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19 Date , 1996.
CSR Number C-7845

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