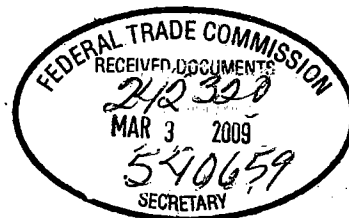


UNITED STATES OF AMERICA
BEFORE THE FEDERAL TRADE COMMISSION
OFFICE OF ADMINISTRATIVE LAW JUDGES



In the Matter of)
)

DANIEL CHAPTER ONE,)
a corporation, and)

JAMES FEIJO,)
individually, and as an officer of)
Daniel Chapter One)
_____)

Docket No. 9329

Public Document

DEPOSITION TESTIMONY SUBMITTED IN SUPPORT OF COMPLAINT
COUNSEL'S MOTION FOR SUMMARY DECISION

In the Matter of:
Daniel Chapter One, et al.

February 6, 2009
Denis R. Miller

Condensed Transcript with Word Index



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1 UNITED STATES DISTRICT COURT
 2 FEDERAL TRADE COMMISSION
 3
 4 In the Matter of:)
 5 DANIEL CHAPTER ONE, a corporation,) Docket No. 9329
 6 and)
 7 JAMES FEIJO, individually, and as)
 8 an officer of Daniel Chapter One,)
 9
 10
 11 Friday, February 6, 2009
 12
 13 Federal Trade Commission
 14 One Bowling Green
 15 New York, New York
 16
 17
 18 The above-entitled matter came on for
 19 deposition, pursuant to Agreement, at 9:30 a.m.
 20
 21 Pages 1 - 194
 22 Reported by: Linda A. Schilt
 23
 24
 25

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1 APPEARANCES:
 2
 3 ON BEHALF OF THE FEDERAL TRADE COMMISSION:
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 5 CAROLE A. PAYNTER, ESQ.
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 7 New York, New York 10004
 8
 9
 10 ON BEHALF OF THE DEFENDANTS:
 11 JAMES S. TURNER, ESQ.
 12 CHRISTOPHER TURNER, ESQ.
 13 BETSY E. LEHRFELD, ESQ.
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 15 1499 16th Street, N.W.
 16 Washington, D.C. 20036
 17
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 21
 22
 23
 24
 25

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1 DR. DENIS R. MILLER, having first been
 2 duly sworn by a Notary Public of the State of New York,
 3 was examined and testified as follows:
 4 EXAMINATION BY
 5 MR. S. TURNER:
 6 Q. Good morning.
 7 A. Good morning.
 8 Q. Dr. Miller, could you state your name, address
 9 and professional title for the record.
 10 A. Yes. Denis R. Miller, D-E-N-I-S. My address
 11 is 36 East Lake Road, Tuxedo Park, New York 10987.
 12 My official title?
 13 Q. Yes, whatever your professional title is.
 14 A. I'm a therapeutic area leader for oncology
 15 hematology at Parexel, P-A-R-E-X-E-L, all capital
 16 letters, International.
 17 Q. Thank you. Dr. Miller, you met Betsy Lehrfeld
 18 who is here, Chris Turner, and I'm Jim Turner, and we
 19 are representing the respondent in this case, Daniel
 20 Chapter One.
 21 A. Yes.
 22 MR. J. TURNER: What we're planning to do today
 23 is go over your expert witness report and talk about
 24 that and I want to do three things: One is to talk
 25 about how the report was prepared, that's the first

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1 part; and the second part is to go through the report
 2 itself; and then the third part is any leftover general
 3 questions or concepts, stuff that we didn't cover in
 4 the previous two sessions. We'll take probably all day
 5 to do this, basically from now until five. I guess
 6 we'll break for lunch for about an hour, 45-minutes to
 7 an hour, right in the neighborhood.
 8 MR. PAYNTER: That sounds fine.
 9 MR. J. TURNER: Whatever makes sense, probably
 10 around noon. If you have any need for a break at any
 11 time, just say I need a break. If you need water,
 12 anything like that, just say you need that, whatever,
 13 and we'll do the same if I have to stop for a while.
 14 We might take a break in the morning sometime and in
 15 the afternoon, you know, for a few minutes. That's
 16 kind of the way we've been doing it.
 17 MR. PAYNTER: Just for the record, Dr. Miller
 18 has an appointment for 7 o'clock this evening.
 19 MR. J. TURNER: I'm reasonably sure I'll be
 20 done by five. That's kind of what we agreed to. It
 21 may go over a little more, it may end before that. I
 22 know what I need to know and when we get there we'll
 23 get there. I'm pretty sure it's not going to go past
 24 five or maybe shortly after five.
 25 MR. PAYNTER: Okay.

1 Q. I wanted to begin, Dr. Miller, with asking you
2 questions about how the report was prepared. So the
3 first question I have is how did you hear about this
4 case?

5 A. I believe I received a telephone call from
6 Mr. Zang, who's not here.

7 MR. PAYNTER: He's here.

8 A. There he is, I'm sorry.

9 And there may have been someone else on the
10 call at that time. I'm not sure if Carole was on the
11 call. I got a call from the FTC.

12 MR. J. TURNER: Are you saying, yes, you were?

13 MR. PAYNTER: I don't know if I was.

14 A. I know Ted was on the call and it was an
15 introductory call broadly finding out who I was and
16 what I had done and whether I had done any work on
17 issues relating to claims about the anticancer activity
18 of certain products.

19 And I reviewed my experience and we had a few
20 more teleconferences where after I had submitted my CV,
21 and it was at that point in time after I signed a
22 confidentiality agreement and a contract was set into
23 place I was then specifically asked to review whether
24 these four products of Daniel Chapter One would satisfy
25 some of the claims that were made about them and

1 claims made that these products all by themselves had
2 potent and effective anticancer activity.

3 Q. Now, I asked you before this answer that you
4 gave what was your understanding the products were,
5 what did you think they were?

6 A. Well, there were four products.

7 Q. What I mean is what class were they; foods,
8 drugs, food additives, what was your understanding?

9 A. Well, I looked at them as agents that would
10 have -- I asked the question do these agents or
11 products have any anticancer activity.

12 Q. How did you come to form that question as the
13 question you were asking or answer?

14 A. It was based upon claims that were made and in
15 support of these four products stating that they could
16 inhibit cancer growth or tumor growth, that they were
17 effective in the treatment of cancer, that they might
18 actually obviate some of the adverse effects of cancer
19 treatment itself.

20 Q. And how did you arrive at those claims as
21 claims that you were going to evaluate?

22 A. From the review of the Daniel Chapter One web
23 site and the supporting information that came from
24 their web site about what their products do and how
25 they might help patients with cancer.

1 whether there was reliable and supportable evidence
2 that these claims were reasonable, scientifically and
3 medically.

4 So then I began my work and that was in October
5 of 2008.

6 Q. And when you were asked about these products,
7 what did you understand the products to be?

8 A. I had to wait until I had gotten the complaint,
9 and I had to wait until I got specific information
10 about the products themselves, and then I began a
11 review of some of the literature and other documents
12 that were submitted by Daniel Chapter One in support of
13 their claims and evidence as well as my own very in
14 depth review of the literature that relates to a number
15 of these compounds or products that have been used in
16 the treatment of cancer.

17 Q. When you say "have been used in the treatment
18 of cancer," what do you mean by that?

19 A. A good example would be shark cartilage. There
20 have been reports of the use of a number of
21 complimentary medicines in its broadest definition that
22 have been used to complement conventional cancer
23 therapy to see whether it might improve quality of life
24 or it may have additive effect to conventional
25 anticancer therapy, and in some cases there have been

1 MR. PAYNTER: Can you read back the question,
2 please.

3 (The requested portion was read.)

4 Q. So now you had in your mind the claims. Had
5 you determined in your mind yet whether you were
6 dealing with a food, a drug, a food additive or some
7 other substance?

8 MR. PAYNTER: I'm just going to object on
9 foundational because you're asking him did he determine
10 the claims and I think you can ask him the question did
11 you determine what the claims were and that might
12 actually clarify it. I think the record is a little
13 unclear right now as to who determined the claims in
14 this case.

15 MR. J. TURNER: Well, actually, I'm going to
16 ask that question more specifically when we get to the
17 claims in the document. What I'm trying to understand
18 and am trying to ascertain is as he began the process
19 what was his assignment.

20 MR. PAYNTER: Well, that might be a better
21 question.

22 A. Well --

23 MR. J. TURNER: That's the generic question.
24 I had already asked that but we can go back through it
25 again.

1 Go ahead.
 2 A. I was asked by the FTC to determine whether
 3 there was competent and reliable scientific evidence to
 4 substantiate a number of claims about these four
 5 products; whether they inhibited tumor growth, whether
 6 they were effective in the treatment of cancer, whether
 7 they can actually eliminate tumors or whether they can
 8 actually heal or obviate the adverse effects or
 9 destructive effects of radiation therapy or
 10 chemotherapy. And I was asked to provide reliable and
 11 competent evidence, if I could find it, in support of
 12 these claims.

13 Q. Was this before or after you saw the complaint?

14 A. Was what before or after I saw the complaint?

15 Q. Had you looked at the web site and formulated
 16 some ideas about claims and had you begun your work and
 17 the question I'm asking is: Did that activity that you
 18 described, and there were some other things in there,
 19 take place before or after you read the FTC complaint?

20 A. I can't tell you exactly the order of things.
 21 There were so many different things that I reviewed.
 22 The complaint was one thing to get a focus on what the
 23 case was all about, but I reviewed all the literature
 24 that was provided by Daniel Chapter One in support of
 25 their position. I reviewed my own literature sources

1 that related to the same issues. I reviewed different
 2 web sites. I reviewed material from different cancer
 3 centers. I reviewed my own huge body of literature in
 4 this area because I've done a lot of work in it. So
 5 there were so many different sources that I reviewed
 6 before I even began writing my report or formalizing my
 7 opinions.

8 Q. I just want to understand. You don't recall
 9 whether you had seen the complaint before you started
 10 the process?

11 MR. PAYNTER: Objection.

12 A. I don't remember.

13 MR. PAYNTER: Objection.

14 MR. J. TURNER: On what ground?

15 MR. GREENE: That's a very unclear question.

16 Q. The question is that you said you began your
 17 activities in October, that's what you recalled?

18 A. Yes.

19 Q. Let's walk through it. Then you did a number
 20 of things that you laid out and described. When did
 21 you begin to do the work that ended up with the report?

22 A. When did I begin my work that related to my
 23 report? In October when I began a review of
 24 everything relating to these products.

25 Q. Do you have any idea when you received a copy

1 of the complaint?

2 A. I don't recall. I listed all the things that I
 3 reviewed but I didn't put down the date I reviewed all
 4 of them because it was an ongoing dynamic process.

5 Q. Okay. What was your reason for taking this
 6 assignment on?

7 A. What was my reason for taking the assignment
 8 on?

9 Q. Yes.

10 A. I'm an oncologist. I spent my career in
 11 treating, diagnosing and I think making some advances
 12 in the way we treat cancer patients, and I'm interested
 13 in all potentially effective therapies to improve the
 14 life of a cancer patient; and I've been doing that all
 15 my life. I've also done a lot of work in what I would
 16 call complimentary medicine, supportive care in cancer
 17 patients. And when I was asked to review this, it was
 18 something I had knowledge of and an interest in and
 19 said, yes, I'd be happy to review these products and
 20 see whether there is competent and reliable evidence to
 21 support their use in treating cancer.

22 Q. Um --

23 A. I never heard of them before and so it was --
 24 except for shark cartilage, but I never heard of this
 25 company before, nor had I heard of any of their

1 products.

2 Q. What are your thoughts about the company,
 3 having done this review, what is your impression of the
 4 company?

5 A. My impression of the company or my impression
 6 of the company doing the review? I'm not sure which
 7 part of that --

8 Q. You reviewed products of a company.

9 A. Yes.

10 Q. What are your impressions of the company?

11 A. I don't know how to answer that, okay.

12 Q. Okay.

13 A. I never met the people who own the company.
 14 All I've read is what they have in the public domain
 15 and that's all I know about them, and I read the
 16 depositions of Jim Feijo and his wife Patricia, Tricia.

17 Q. Okay.

18 A. That's all I know about the company, but I
 19 never met them personally, never interviewed them,
 20 never visited their sites of business.

21 Q. I want to now go to the second part of this,
 22 which is the main activity here, which is going over
 23 the report itself. We've done a little bit of that now
 24 because you used some of it to answer these questions
 25 but we may go over some of that.

1 Do you have a background in nutrition?
 2 A. Am I a nutritionist, no. Do I know about
 3 nutrition as it relates to cancer patients, yes.
 4 Q. Can you describe your knowledge about nutrition
 5 as it relates to cancer patients?
 6 A. Well, I'm very aware of the importance of
 7 nutrition in cancer patients. I'm very well aware of
 8 the adverse effects of malnutrition. I'm aware of how
 9 important it is for cancer patients who are undergoing
 10 therapy to make sure that they're well hydrated and not
 11 malnourished and, if they are, to treat those
 12 deficiencies so they can tolerate their treatment
 13 better and have a better quality of life.
 14 I am constantly engaged in working with
 15 nutritionists and metabolic colleagues to help support
 16 cancer patients that I treated in a comprehensive and
 17 full way.
 18 Q. Do you have any training in nutrition?
 19 A. No.
 20 Q. Do you have any certifications in nutrition?
 21 A. No.
 22 Q. I noted in your credentials that you were
 23 involved in oncology/hematology. Is that your area of
 24 expertise?
 25 A. I'm board certified in oncology and hematology.

1 into board certification in either oncology or
 2 hematology. Some people have one or the other and some
 3 people have both. In pediatrics it's a combined board
 4 certification.
 5 Q. When you're certified in oncology/hematology
 6 you're certified in all oncology?
 7 A. Yes.
 8 Q. All tumors and not just blood?
 9 A. No. Oncology covers all cancer and, as I said,
 10 some hematologic malignancies are also cancer.
 11 Leukemia is a cancer of the blood. Hematology goes
 12 beyond cancer. It includes things like anemia. It
 13 could include things like bleeding disorders, like
 14 hemophilia. It includes clotting disorders for people
 15 who develop blood clots. It might include
 16 non-malignant disorders that effect any of the
 17 different blood cells of the body.
 18 Q. Does leukemia involve tumors?
 19 A. Leukemia is a hematologic malignancy that is
 20 not considered a solid tumor. Blood malignancies are
 21 not the same as a colon cancer. There is nothing solid
 22 about leukemia.
 23 Q. When you're certified in oncology/hematology,
 24 you would be pediatric oncology/hematology, that is
 25 what your certification is in?

1 Q. Do you have other board certifications?
 2 A. Pediatrics.
 3 Q. Could you describe what oncology/hematology is?
 4 A. Oncology is the study of the diagnosis, cause,
 5 treatment of cancer.
 6 And hematology is the study of the cause,
 7 diagnosis and treatment of blood diseases. Some blood
 8 diseases are cancers.
 9 Q. Do they involve tumors?
 10 A. Yes.
 11 Q. A blood disease -- does blood oncology involve
 12 tumors?
 13 A. Blood tumors.
 14 Q. Oncology/hematology, does that involve tumors?
 15 A. Oncology is cancer, which can include solid
 16 tumors and disorders like leukemia or lymphoma which
 17 are hematologic malignancies.
 18 Q. What is your board certification in?
 19 A. Pediatrics and pediatric hematology/oncology.
 20 Q. In hematology/oncology, that's two things; one
 21 is hematology and the other is oncology.
 22 A. In pediatric board certification you get
 23 certification for both oncology and hematology.
 24 Q. Go ahead.
 25 A. In medicine, internal medicine, it's divided

1 A. Yes.
 2 Q. I want to understand, just to clarify. You
 3 originally said you were certified in pediatrics and
 4 that you were certified in oncology/hematology. Is
 5 that two separate certifications or one combined
 6 certification?
 7 A. One has to be trained in general pediatrics
 8 first, and then gets additional training in hematology
 9 and oncology to qualify for certification in hematology
 10 and oncology.
 11 Q. If someone is qualifying for oncology and
 12 hematology, do they have to have a certification in
 13 pediatrics?
 14 A. I didn't understand that.
 15 Q. If a person is seeking certification in
 16 oncology/hematology, do they need to be certified in
 17 pediatrics first?
 18 A. If it's pediatric hematology/oncology that
 19 they're going for, is that what you mean?
 20 Q. No. I'm just going by what it says here. Are
 21 you certified in pediatric oncology/hematology?
 22 A. Yes. Let me just clarify because it's very
 23 confusing for anybody trying to read this. You have to
 24 be certified in pediatrics first. That means you have
 25 to complete a residency in pediatrics. Once you've

1 done that, then you go on and take a fellowship in
2 oncology/hematology in pediatrics, and after
3 successfully completing your fellowship training, and
4 successfully passing the board examination, you then
5 become certified in hematology/oncology combined in
6 pediatrics.

7 Q. And that would certify you to be qualified to
8 do colon cancer, pediatric colon cancer?

9 A. Well, if indeed I saw a case of pediatric colon
10 cancer, and I have, yes, I'll be certified to do that.

11 Q. That's what I'm trying to get at. I had
12 skipped a paragraph.

13 You have been involved with a number of
14 institutions, University of Rochester Medical Center,
15 New York-Cornell Medical Center, Memorial Sloan
16 Kettering and Northwestern University Medical School;
17 is that right?

18 A. That's correct.

19 Q. How were you funded in those jobs? Were you
20 paid by those institutions?

21 A. I was paid by those institutions, correct.

22 Q. Did you have grants from any sources?

23 A. Yes, I did have grants that supported my
24 research work at those institutions.

25 Q. Can you tell me where those grants came from?

1 A. At Rochester Medical Center, New York
2 Hospital-Cornell, Memorial Sloan Kettering and at
3 Northwestern most of the grants came from the National
4 Cancer Institute

5 Q. How about the Cornell, same?

6 A. Well, Cornell is New York Hospital Medical
7 Center. Yes, the grants I had then came primarily from
8 the National Cancer Institute. At New York
9 Hospital-Cornell, our department, our division in
10 hematology/oncology was funded by a private
11 philanthropic organization, Children's Blood
12 Foundation, which is here in New York City, which
13 provided a large portion of the support for the whole
14 division. Salaries for the faculty, research program,
15 fellowship program and the funds went to the
16 university, to the medical school, but the research
17 foundation funded a great deal of what we were doing at
18 New York Hospital-Cornell.

19 At Memorial Sloan Kettering I had a large
20 program project grant from the National Cancer
21 Institute to study hematologic malignancies.

22 Q. Do hematologic malignancies involve tumors?

23 A. You asked me that question. I'll try to
24 explain it. When you think of a tumor, think of a
25 breast cancer, think of a brain tumor or think of

1 pancreatic cancer. They're solid tumors.

2 When you think of a blood tumor, malignancy of
3 the blood, hematologic malignancy, think of a cell
4 floating around the body in the blood stream or lymph
5 nodes. So they're not solid tumors, if you will,
6 they're liquid tumors. They're still cancer but it's
7 just what kind of cancer it is.

8 Q. In your practice you worked on both solid
9 tumors and liquid tumors that you just called them?

10 A. Yes.

11 Q. What is the ratio of solid tumor work you've
12 done versus liquid tumor?

13 A. Depends what part of my career.

14 Q. How about while you were working at these
15 institutions?

16 A. Up until 1990 when I had positions as either
17 chairman of a department or division head in a
18 hematology/oncology program, most of my own clinical
19 activities and my own research activities involved
20 hematologic malignancies, leukemia, although I took
21 care of patients with solid tumors, brain soft tissue
22 sarcomas or any of the solid tumors we saw in
23 pediatrics.

24 In 1990 I had a major career shift and at that
25 time joined an organization that was involved primarily

1 in the diagnosis and treatment of adult patients with
2 cancer. So that from 1990 until today, most of my
3 clinical activities involve tumors that are seen in
4 adult population more commonly than in pediatric
5 population.

6 Q. Those are more commonly solid tumors?

7 A. More commonly solid tumors, although I'm still
8 doing work with hematologic malignancies.

9 Q. You described this now as the treatment of
10 patients?

11 A. Diagnosis and treatment.

12 Q. And treatment. With regard to your research
13 activity, was it pretty much the same ratio and the
14 same experience in your career change?

15 A. Again, before 1990 it was primarily hematologic
16 malignancies and I would say 80 percent was hematologic
17 malignancy in terms of my time and effort in the clinic
18 or laboratory.

19 From 1990 until the present day the activity
20 has been more in solid tumors, like non-small cell lung
21 cancer, breast cancer, colon cancer, although there is
22 activities that I have now that relate to lymphomas and
23 leukemias, but it's more solid tumors because of the
24 adult population. Solid tumors are more common than
25 hematologic malignancy.

1 Q. You said in 1990 you had a major career change.
2 What was that career change?

3 A. I left an academic environment in a teaching
4 hospital and became the associate medical director of
5 an organization called Cancer Treatment Centers of
6 America, so I was the associate medical director there.
7 And I also was in charge of the clinic research program
8 at the different hospitals, centers and clinics of
9 Cancer Treatment Centers of America.

10 In 1993 I became the scientific director of the
11 not-for-profit research activity in Cancer Treatment
12 Centers of America called Cancer Treatment Research
13 Foundation. I still had my clinical activities at the
14 hospital and even during that time I had my own
15 clinical activities taking care of children and
16 adolescents with cancer, but my work shifted in terms
17 of actually directing the clinical research program
18 inpatients with adult patients with cancer, which meant
19 I helped in my own protocol development, brought in new
20 agents to evaluate patients with advanced stage cancer.
21 These were agents that were undergoing clinical
22 investigation and had not yet been approved. And we
23 also were involved in a very broad program of providing
24 total comprehensive care to patients.

25 Q. Can you describe what total comprehensive care

1 willing to give up. They're willing to try something
2 that might be effective that might prolong their lives
3 to get them from Thanksgiving through the new year.

4 So many of the patients that came were either
5 referred by other doctors or came as several referrals
6 of patients with very advanced stage disease and in
7 some cases we could offer those patients additional
8 therapies. I'm talking about conventional therapies,
9 or an investigational therapy they were interested in
10 participating in, clinical trial.

11 At the same time we were very tuned into
12 looking at the patient's nutrition, looking at other
13 deficiencies the patient might have, looking to see
14 whether there were psychosocial issues that were
15 impacting on their ability to tolerate therapy, were
16 they depressed, do they need psychosocial support. All
17 of those were part of the total comprehensive care the
18 patients got.

19 Q. What kind of criteria did you use to decide if
20 somebody said I don't want to give up and get my
21 affairs in order, I want to go from Thanksgiving to
22 Christmas, what kind of criteria do you use to assign
23 things to them?

24 A. Well, first of all, if you're going to put a
25 patient on a clinical trial, clinical study, you want

1 involves?

2 A. Patient has cancer, it has to be diagnosed and
3 treated effectively, but patients with cancer have
4 other needs. They have psychosocial problems, may have
5 nutritional problems. They need good supportive care
6 so the philosophy at Cancer Treatment Centers of
7 America was to provide total comprehensive care to
8 cancer patients to bring in not only cancer doctors but
9 nutritionists, psychosocial support people, other
10 members of the team that would improve the overall
11 therapy of the patient with cancer.

12 Q. What would the typical patient that comes to
13 American Cancer Centers -- is that it?

14 A. Cancer Treatment Centers of America.

15 Q. When they arrive there, what kind of program
16 would they be put into, treated as?

17 A. Depends on the patient. Most of these patients
18 were previously treated who had one or more recurrences
19 of their disease. Often they came because at their own
20 hospitals or in the clinics where they were being
21 treated, their advice was not too much more we can do
22 for you, your disease has been through all the
23 available therapies, you may want to just consider
24 quality of life, no more treatment and get your affairs
25 in order. And patients, many patients today are not

1 to make sure that the patient meets certain eligibility
2 criteria. If they're in congestive heart failure and
3 their liver is failed and kidneys aren't working,
4 they're not going to be able to tolerate treatment very
5 well. So you want to make sure that patients meet
6 rather straightforward and important criteria that
7 would make them eligible for the study, one of which
8 would be what is their estimated lifespan. If a
9 patient is so far advanced in the disease and the
10 disease has effected vital organs in the body, like the
11 liver or the heart or the lungs or kidneys, those
12 patients are not going to tolerate therapy very well so
13 you'll never be able to test whether a new treatment is
14 effective or not.

15 Q. What do you do with those patients?

16 A. We give them our advice about what we think
17 might be best for them. Some of those patients are not
18 considered candidates for treatment but they're given
19 supportive care.

20 Q. What kind of supportive care would you --

21 A. Well, if the patient is depressed, they might
22 need psychosocial, psychiatric support. If they're
23 malnourished, they could be treated with nutritional
24 support if they wanted it. If they have serious pain
25 problems, they could be given better coverage for their

1 pain because cancer pain is a major problem. Those are
2 the kinds of things that we would look at.

3 Q. What role does their desire play in your
4 treatment prescribed for them?

5 A. It's absolute. The patient has to provide you
6 with informed consent to go on any treatment and the
7 patient has to be a partner in that treatment program.
8 You can't force anything on somebody. They have some
9 empowerment. Yes, I want to go along with that
10 program, or no, I don't.

11 Q. Now, I understand from what you're saying that
12 some people who come there, even in the conditions that
13 they are, are treated with conventional
14 chemotherapeutic agents; is that right?

15 A. Depends on what their prior therapy has been.
16 Some patients may have been through all the
17 conventional hemotherapeutic agents, including
18 radiation and surgery, conventional therapeutic agents
19 and are maybe no longer responding to any of them. And
20 patients like that might be candidates for a study
21 that's looking at a new investigational drug at a much
22 earlier stage in the development. It may be
23 chemotherapy or what we call targeted therapy, going
24 after some unique feature of the cancer itself, and
25 these are early phase studies where we don't -- these

1 are not approved drugs. They've gone through a certain
2 process of evaluation before they ever were used in a
3 human being with cancer, but in some of these studies
4 we were just trying to determine what the most
5 effective dose might be to move on to seeing whether
6 it's going to be active against specific types of
7 cancer.

8 Q. I want to continue asking you questions about
9 what we just have been discussing, but I want to --
10 before I do that -- ask you some background questions.
11 How long did you remain at the cancer center?

12 A. I was at Cancer Treatment Centers of America
13 and the Cancer Treatment Research Foundation from 1990
14 until the end of 1996.

15 Q. Then what did you do career wise at that point?

16 A. I moved from the Chicago area back home, which
17 is the Metropolitan New York area, and actually joined
18 a start-up biotech company developing a new innovative
19 therapy for the treatment of cancer. I was their vice
20 president for clinical oncology.

21 Q. How long did you remain there?

22 A. Until the company went belly up, which was
23 about eight months later.

24 Q. Eight months later?

25 A. Yes.

1 Q. What did you do at that point?

2 A. At that point I had a choice of going back into
3 academia or actually going into the pharmaceutical
4 industry or doing my own thing, and what I did was my
5 own thing. I created my own consulting company, one
6 chief, that was me, no Indians, and I worked with the
7 pharmaceutical industry in areas of my expertise to
8 help them in their development of primarily new agents
9 to treat cancer or blood diseases.

10 Q. What was the name of the organization?

11 A. Expert Medical Consultants, Inc.

12 Q. How long did you maintain that entity?

13 A. Well, I still maintain it but only for
14 activities like this. I'm full-time in the job I have
15 and I've been full-time in the industry since about
16 2003, but during that time --

17 Q. You said full-time in --

18 A. In industry.

19 Q. What do you mean by "industry"?

20 A. Either the pharmaceutical industry or with a
21 contract research organization.

22 Q. Is that a particular organization that you were
23 with?

24 A. Well, maybe we should go through my CV so it's
25 clear. I worked with a number of different

1 organizations when I had my company called Expert
2 Medical Consultants. I work with, for example, a
3 company in New Jersey that was developing a new drug to
4 treat pancreatic cancer and mesothelioma, which is the
5 wall of the peritoneal cavity or pleural cavity. So I
6 worked part-time with them, helping them with their
7 clinical development program, interaction with the FDA.
8 I wrote some of their study reports and helped them
9 move their drug along.

10 At the same time I worked with another company
11 out in California that was developing a drug to treat
12 tumors that were pretty superficial where if you gave a
13 certain drug intravenously, it would be picked up by
14 the tumor in the tumor cells, and if you hit that tumor
15 with a certain wavelength, laser therapy, you could
16 cause a reaction inside the tumor that would result in
17 the destruction of the tumor cells, photodynamic
18 therapy. And a company out in California was
19 developing both the laser and the drug to treat
20 superficial cancers, like skin cancer, bladder cancer,
21 lung cancer, that could be reached by a tube that you
22 can put down the windpipe and into the major airway
23 passages in the lung.

24 I also worked with a contract research
25 organization at that time and was a medical monitor

1 managing one of their large clinical trials that they
2 were helping another pharmaceutical company conduct.
3 Small companies don't have the resources to do all
4 this, so they contact out to what is called a contract
5 research organization to do all of that study
6 management for them.

7 That was a drug that was being looked at in the
8 treatment of myeloid leukemia and malignant melanoma.
9 I also worked with the company I'm currently working
10 with as a medical monitor and I, as a consultant,
11 managed a huge study of a new targeted therapy that was
12 designed to treat non-small cell lung cancer. It was
13 something that could be given by mouth. It was
14 absorbed by the body. It was currently in phase II,
15 III to see whether it was effective in the treatment of
16 lung cancer patients who were on chemotherapy or could
17 it be used alone on inpatients who have been through a
18 number of different lines of treatment for their
19 disease.

20 Serving as a medical monitor on this study, I
21 interacted with the different oncologists around the
22 county who was entering patients on the study, answered
23 questions about eligibility and made sure there were no
24 safety issues that needed to be looked at more
25 vigilantly and made sure they were getting the drugs

1 anemia associated with chemotherapy.
2 I've been with PAREXEL since 2006, January 2006
3 as a therapeutic area leader for oncology and
4 hematology.

5 To summarize, since 1990 I would say that
6 95 percent of the studies that I have been involved in
7 as well as the drugs I've helped develop or the
8 supportive care drugs that I worked on have been
9 inpatients over the age of 18. I'm board certified in
10 hematology/oncology pediatrics but for the last
11 18 years my professional career has been basically
12 involved in understanding cancer in adult patients,
13 designing treatment programs for those patients and
14 evaluating the results of those treatment programs and
15 understanding more about their diseases and better ways
16 to treat them.

17 Q. During that time have you been also continuing
18 to treat patients?

19 A. I stopped any kind of patient care activities
20 in 1996.

21 Q. So from '96 --

22 A. I don't have any direct hands-on care
23 activities since 1996.

24 Q. What is a medical monitor?

25 A. A medical monitor is a physician trained in

1 that they needed to treat their patients.

2 While I was doing that as a consultant, I was
3 also doing consulting work for Hoffman LaRoche and at
4 that time was working on the development and eventual
5 approval of a brand new drug that was developed to
6 treat lymphoma, a real breakthrough, because that drug
7 when given with chemotherapy and for the first time in
8 about 25 years it really improved response rates, the
9 remission duration rates as well as survival of
10 patients with non-Hodgkin, H-O-D-G-K-I-N, lymphoma.

11 So I was involved in the whole process of
12 completing those clinical trials and helping get that
13 drug approved primarily in Europe first before it got
14 approved in the United States. It got approved in the
15 United States three years later.

16 Then I became full-time at Hoffman LaRoche in
17 about 2003 I think and was working on the lymphoma
18 project but also was working on another area of great
19 interest, and that was the use of an agent that is
20 actually a mimic of the same hormone our body produces
21 to help the body make red blood cells to treat the
22 anemia that is caused by the chemotherapy. I helped
23 that drug.

24 In 2004 I moved to Johnson and Johnson where I
25 was working on that same class of agents to treat the

1 oncology. For example, if it's a cancer study, who is
2 available to interact with the doctors at the clinics,
3 at the hospital who are actually treating their
4 patients on a particular clinical study. There are
5 questions that come up about whether a patient might be
6 eligible for the study, does the patient meet the
7 eligibility criteria for this drug in this indication,
8 do they have a specific diagnosis, do they have that
9 stage of disease, how many kinds of prior therapies
10 have they had, is their clinical condition adequate,
11 are the available tissues there for review. All of
12 those things are major questions, eligible questions
13 that come up all the time.

14 There is a lot of interaction with study nurse
15 coordinators that work with the oncologist at a
16 particular clinic or cancer hospital who may have
17 questions about the administration of the new drug
18 intravenously or maybe a better way to keep it stored.

19 Other things that come up are safety issues, a
20 patient has some adverse effect of treatment and there
21 was a question of whether it was caused by a new drug
22 or whether it was part of the disease.

23 The medical monitor also reviews a lot of the
24 safety reports. If a patient has some kind of adverse
25 event and it is a serious adverse event, a report has

1 to be filled out promptly and a determination has to be
2 made about whether that adverse event is related to the
3 drug or not related to the drug because if it is, a
4 report has to be sent in to the FDA. Other
5 investigators using that drug have to be alerted to the
6 fact. So that is a major role of a medical monitor is
7 to evaluate safety.

8 The monitor also looks at some of the
9 laboratory data coming in to make sure things are not
10 alarming or off the charts that might be related to the
11 drug itself.

12 Q. You had indicated that in one of your
13 positions, I guess Hoffman LaRoche, you came up with
14 something for the first time in 25 years that effected
15 various rates?

16 A. Yes.

17 Q. Tell me about the response rate. How did it
18 effect the response rate?

19 A. It improved it. The study was taking
20 conventional chemotherapy for the treatment of
21 non-Hodgkin lymphoma, which was -- had been used for
22 25 years, variations of it had to be used, attempts to
23 make it more toxic or more intense weren't better and
24 in the '90s people were available to develop a
25 monoclonal antibody. This monoclonal antibody, think

1 of it as a missile targeted to a specific target on the
2 lymphoma cell. This monoclonal antibody would
3 actually identify this target on the lymphoma cell,
4 attach to it and then set into motion a series of
5 events that would cause the destruction of that tumor
6 cell. And it was really like a targeted missile that
7 would effect that tumor cell rather than normal cells.
8 In a controlled trial patients were either given the
9 standard therapy or they were given the standard
10 therapy plus this monoclonal antibody, and the
11 response rates were statistically significantly better
12 because the numbers were large enough to show there was
13 a statistically chance improvement in the response
14 rate. The duration of that response in the patients
15 getting the monoclonal antibody and chemotherapy were
16 significantly better and the overall survival was
17 significantly better in the patients receiving
18 combination therapy monoclonal antibody.

19 Q. When you say "significantly better" what are
20 the rates we're talking about?

21 A. Response rates of over 75, 80 percent,
22 five-year survivals. Now it is even a seven-year
23 survival because recent update on the study is in the
24 range of 65 percent, and if you've survived lymphoma
25 for two years or more after your treatment has been

1 discontinued, chances are it's not going to come back
2 again.

3 Q. What was the difference between the treated
4 group and the controlled group?

5 A. 10 or 15 percent.

6 Q. So these were randomly?

7 A. Yes.

8 Q. So the people randomly assigned the new product
9 had a 15 percent better chance of surviving?

10 A. That's right.

11 Q. When I asked you about response rate -- and I
12 gather we just discussed survival rate?

13 A. I talked about the five-year survival rate. I
14 think I mentioned a number for the response rate. I
15 would really prefer to look at the document to give you
16 the exact numbers. I don't want to do something from
17 memory.

18 When I say there was a statistically
19 significant improvement in response rate, that's again
20 based on numbers of patients empowering the difference,
21 it's not by chance, and response is clearly evaluated.
22 It's not I feel better, gee, my tumor went away. It's
23 demonstration that there is no tumor based on physical
24 exam, medical imaging studies. That's what's needed to
25 quantify a response. You can tell how long the

1 response lasts by measuring the time from when it
2 occurred to when the disease comes back again. So we
3 have another measure, very important time to tumor
4 progress, or time to disease progression and that was
5 significantly better in the patient who got the
6 chemotherapy plus the monoclonal antibody. And the
7 same is true in a study that's been followed for over
8 seven years, which is a long time for a study.

9 So each one of those major end points,
10 response, but more important is survival, that is the
11 key thing, did you live or not, and survival was
12 significantly better.

13 Q. That goes for remission as well?

14 A. Remission was better. More important, a lot of
15 people go into remission but it doesn't last long and
16 the disease comes back. They get treated some other
17 kind of treatment. They go into remission but it
18 doesn't last long and often the second time around it
19 lasts shorter. These are patients who have never been
20 treated before and their response rates were better in
21 the group who received chemotherapy and monoclonal
22 antibody. Their time to tumor progression was longer
23 significantly and proportion of patients alive after
24 five, seven years was significantly higher in that
25 group.

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1 Q. How do people qualify to be in or out of such a
2 study?
3 A. For that particular study they had to have a
4 certain kind of non-Hodgkin lymphoma. It was the
5 aggressive kind. It had to be a lymphoma that
6 expressed the target of the monoclonal antibody. They
7 had to have a B cell lymphoma and they had to meet the
8 other eligibility criteria of the study relating to the
9 age, physical examination, organ function and of course
10 they had to provide consent to go on to the study.
11 Q. What happened to the people who didn't qualify
12 for the study?
13 A. They got treated some other kind of therapy for
14 non-Hodgkin lymphoma. Some patients wish not to go on
15 a clinical trial. Medical oncology, 90 percent,
16 95 percent of patients don't want to be enrolled in a
17 clinical trial.
18 Q. Why is that?
19 A. They want to get something that is going to be
20 effective. They don't want to be randomized perhaps
21 placebo. They don't want to have to travel to a major
22 cancer center with all of the inconvenience.
23 It's interesting in pediatric oncology. It's
24 reverse, 95 to 100 percent of children are enrolled in
25 a cancer center or international trial.

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1 Q. What is the difference?
2 A. Parents have a greater control over their
3 children and are responsible for them. An individual
4 may or may not wish to have any kind of treatment.
5 Q. How do the survival and remission and response
6 rates in the pediatric trials compare to those in the
7 adult trials?
8 A. Again, it would depend on what tumor you're
9 talking about. I can't give you a broad number for all
10 pediatric cancer. It includes many, many different
11 types of cancer, so if you would like to ask me about a
12 particular type of cancer, I'd be happy to address
13 that.
14 Q. Let's take Hodgkin lymphoma.
15 A. That isn't what I was talking about.
16 Q. What were you talking about?
17 A. Non-Hodgkin lymphoma.
18 Let me take acute lymphoblastic leukemia. I
19 would pick that because it is the most common
20 malignancy in children, 35, 30 to 35 percent of cancer
21 in children. Today's chemotherapy, the complete
22 remission rates are over 95 to 98 percent. The
23 patients who are alive and well and without relapse of
24 their leukemia three years later depends a little bit
25 on some of the disease factors or patient factors, but

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1 overall the cure rate of acute lymphoblastic leukemia
2 today is 80 percent. Some patients do better than
3 that.
4 Q. Is that unique for various types of cancers?
5 Is that a high rate or low?
6 A. Very high rate. There are Hodgkin diseases
7 that have a cure rate of 90 percent in children.
8 Certain solid tumors in children, like kidney tumors,
9 also have a very high cure rate. But there are other
10 tumor types that have been more difficult to cure,
11 certain bone tumors, certain tumors of the central
12 nervous system, certain brain tumors. So it's not
13 uniform, but acute lymphoblastic leukemia I think is
14 the model that we use to show that with clinical
15 trials, clinical research, learning more about the
16 biology of the disease, understanding what causes it,
17 going after specific targets of the disease,
18 understanding that not all patients with lymphoblastic
19 leukemia are the same. Some patients don't need as
20 much aggressive therapy as others, so you can minimize
21 the toxicity, maximize the efficacy and decrease a lot
22 of the toxic effects of therapy.
23 And I have been involved in a lot of studies
24 and there are other patients who may need more
25 aggressive therapy if you have a chance to cure their

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1 disease.
2 Q. Is pediatric --
3 MR. J. TURNER: Let me try to approach it this
4 way.
5 Q. The field of pediatric oncology, does it have
6 the reputation of being generally more successful in
7 the treatment it provides than the general level of
8 cancer treatments?
9 A. Generally as a general statement that's true.
10 Part of it relates to the nature of tumors in children
11 compared to adults. Lymphoblastic leukemia is much
12 more responsive to treatment than pancreatic cancer is.
13 Fortunately we don't see pancreatic cancer in children.
14 It's the nature of the tumor and available therapies we
15 have for it. Tumors are very responsive and others
16 don't respond at all. You can't cut out leukemia. You
17 can't do surgery on lymphoma unless it is a unique
18 unusual circumstance, but you can't go after all the
19 leukemia cells in the body which may measure, if you
20 like numbers, maybe at the time of diagnosis there are
21 10 to 11th power, okay, ten to the 11th power tumor
22 cells.
23 Q. That's when it starts to manifest itself?
24 A. That's when it manifests.
25 Q. When it's ten to the fifth power --

1 A. You're in remission.
 2 Q. What if you haven't had any that expressed
 3 itself yet?
 4 A. It would be very -- it's at the level of
 5 detection by going into the bone marrow or the blood
 6 and getting cells and then doing very special tests to
 7 see whether you can see the leukemic clone of cells.
 8 That would be the level of detection.
 9 Q. So maybe ten to the fourth you might?
 10 A. Trouble.
 11 Q. Trouble?
 12 A. Trouble.
 13 Q. Is there anything that can be done for people
 14 when they're at ten to the fourth or smaller that would
 15 help them not go to ten to the 11th?
 16 A. We're just learning about what we call minimal
 17 residual disease in patients who have been treated to
 18 see if we get the number of leukemic cells down to that
 19 lower level.
 20 Q. If you had them up and were bringing them down?
 21 A. We bring them down. We don't go in and do bone
 22 marrows on kids in the third grade just to see if they
 23 have ten to the third.
 24 Q. Before you ever have a manifestation, if you
 25 have somebody who is going to eventually have ten to

1 you're looking for something like polyps?
 2 A. We also know that some patients may be more
 3 susceptible and at higher risks. If a woman's mother
 4 had breast cancer, a small proportion of woman inherit
 5 that breast cancer from their mother and you can look
 6 for that gene that increases your risk of developing
 7 breast cancer.
 8 Q. Let me ask you about these phase studies that
 9 you have described. You had mentioned what you call
 10 phase II and III studies.
 11 A. Yes.
 12 Q. Could you give sort of a brief orienting
 13 summary of each of those?
 14 A. I'd be happy to. There is a little bit of a
 15 preface though because -- I'll limit it to oncology.
 16 Q. Yes. This is limited to oncology.
 17 A. Because there are differences. Before we get
 18 to phase I in oncology, we do what we call non-clinical
 19 studies. They can be done in what we call in vivo,
 20 which means in glass, like a petri dish or test tube
 21 where we take cancer cells, not necessarily from the
 22 patient, but cancer cells and see if certain agents
 23 have activity against them, cause their death and stop
 24 their proliferation. We look at how these new agents
 25 might work in specific metabolic pathways inside the

1 the 11th and they're going to start at ten to the one
 2 and build up; is that right?
 3 A. That can happen but in leukemia that is not a
 4 good model. There are other models to take people at
 5 risk.
 6 Q. How would a model like that work?
 7 A. Someone with a family history of polyps in
 8 their colon, grandfather had polyps and he developed
 9 colon cancer. Gentleman's father also had colon cancer
 10 and had polyps and we know polyps can develop into
 11 colon cancer, so they should have frequent
 12 colonoscopies at an early age and have the polyps
 13 excised and examined under the microscope to make sure
 14 it hasn't turned into a malignancy. We don't take out
 15 his colon, but we follow him carefully.
 16 That's why we do mammographies in women,
 17 because early detection, particularly of solid tumors,
 18 is very important for outcome.
 19 Q. But let me ask this question then. There is a
 20 point at which in this case you said ten to the 11th in
 21 every one of the diseases in cancer has a point which
 22 it can be detected?
 23 A. It's different for all, but correct.
 24 Q. Before that there is a point where the disease
 25 potential can't be detected necessarily. That's when

1 cancer cell. We can take tumor cells and inject them
 2 into mice or other rodents or other animals and treat
 3 them with these new agents to see whether we get
 4 evidence of shrinkage of the tumor or disappearance and
 5 we can look at different doses of the drug, give it in
 6 different ways, intravenously, orally or directly into
 7 the different cavities of the body.
 8 Once from the animal studies we have an idea
 9 about some of the safety features of the drug, what
 10 kind of toxicity does it cause, an idea about how its
 11 metabolized in the animals, about how it's excreted
 12 activity against different type of tumors, we take a
 13 much lower dose that we looked at in the animals and do
 14 what -- we do our first phase I study in cancer
 15 patients.
 16 But because we have active, approved, safe and
 17 effective therapies for cancer patients, we can't take
 18 a previously undiagnosed patient with colorectal cancer
 19 who would be a candidate for chemotherapy and put them
 20 on a phase I study. That is unethical. I don't know
 21 anything about the safety of the drug, I don't know
 22 what the right dose should be and I don't have any
 23 idea, I have no idea about whether it would be
 24 effective in colon cancer.
 25 So in phase I my aim is or our aim is to learn

<p style="text-align: right;">Page 46</p> <p>1 a lot about the safety of the drug and what its side 2 effects are in different tissues and organs of the 3 body, effect on the blood, liver, the heart, lungs, 4 kidneys, GI tract, all of those things are looked at. 5 So safety is one of the most important things we do in 6 phase I. 7 Another thing we do in phase I is to determine 8 what the effective dose is going to be when we move 9 into the next phase of clinical trials. So we start 10 off with low doses and after three or six patients, we 11 move the dose up and move it up again and keep moving 12 up until we get what we call dose limiting toxicity, 13 which means that we've identified certain kind of 14 adverse effects that we will consider limiting in terms 15 of whether we can advance the dose any further. 16 Once we've established that, we determine what 17 we call the maximum tolerated dose and either that or 18 one dose level lower is what's used in the next phase 19 of a study, which we call phase II. In phase II our 20 goal is to see whether the drug at that dose level has 21 activity against either a single cancer type or 22 multiple cancer types. 23 In the phase I all of these patients have been 24 previously treated, they all have measurable disease, 25 they have been diagnosed with cancer. They're not</p>	<p style="text-align: right;">Page 48</p> <p>1 often it's double blind, randomized, controlled trial 2 where everyone is getting the same basic chemotherapy, 3 for example, for non-small cell lung cancer and 4 patients are going to be randomly assigned to either 5 that plus a placebo, standard chemotherapy plus 6 placebo, or standard chemo though brand-new targeted 7 therapy directed against the specific target in the 8 lung cancer cell. 9 On the surface there may be receptors. Think 10 of it as a key in the lock and the key is this new 11 targeted therapy. So we have the lock is the receptor 12 on a non-small cell lung cancer cell and the new drug, 13 which is something you can take by mouth, is directed 14 against that target specifically. And if you don't 15 express the target -- and now we know if you don't 16 express it in a very special way where it's got 17 changes, mutations, that drug isn't going to work. It 18 can be a monoclonal antibody, it can be a small 19 molecule, you can take by both and what you can do then 20 if it's a little pill, some patients can get a placebo, 21 other patients can get a new drug and see what kind of 22 response rates they have, what kind -- 23 Q. This is in phase III? 24 A. This is phase III. Response rates are not as 25 important though, but what really is important is you</p>
<p style="text-align: right;">Page 47</p> <p>1 getting anything else but the experimental agent 2 usually. Sometimes you might give a conventional 3 therapeutic agent, but not often. 4 In phase II once you establish that dose, then 5 you are looking for efficacy, you're looking for a 6 response, tumor shrinkage primarily. You might look at 7 a number of different tumor types, depends on what type 8 of drug it might be and how it works best. If you see 9 evidence of activity in a phase II, you might use it 10 with other conventional therapeutic agents to see 11 whether it is safe and also effective. There sometimes 12 is a way to do a randomized trial in phase II where 13 patients could go on conventional chemotherapy with the 14 new agent versus conventional chemotherapy alone and 15 look for response time to tumor progression. 16 Q. That study that you described for Hoffman 17 LaRoche, that came up with the breakthrough? 18 A. It was a phase III trial. Again, in phase II 19 you can take previously untreated patients, if you're 20 comparing standard therapy alone with standard therapy 21 plus the new agent, that would be reasonable because no 22 one is going to be denied what is the standard of care, 23 but in phase III, often you take the standard of care 24 and in a randomized way, doesn't have to be double 25 blind, but depends on the drug, can be open label, but</p>	<p style="text-align: right;">Page 49</p> <p>1 have prolonged the survival of that patient. You 2 prolong the time from when their diagnosis has been 3 made until their tumor progresses, so these are 4 patients who have advanced stage disease generally. 5 Or also do it in a patient who had surgery, 6 disease is gone, breast cancer, after surgery, they 7 don't have the lump or have their breast but we know 8 that is not enough, so we treat them with additional 9 therapy to prevent the disease from coming back again 10 because there are a few cells we can't see. So a 11 number of different stages of the disease based on the 12 extent of the disease but, again, the end points are in 13 phase III improvement in what we call progression free 14 survival or overall survival, that is what we're 15 looking for. Response rates are not as important in 16 phase III. 17 Q. What does it cost to do these studies? 18 A. From the beginning, from the non-clinical? 19 Q. You have a promising item. 20 A. Let's say you have gone through testing of 100 21 different compounds in the clinic and you see one that 22 might be better, so there is expense there. It may 23 cost upwards of a hundred million dollars to go from 24 the beginning to the time a drug goes through phase 25 III.</p>

1 Q. You mentioned in your report that out of 5,000
2 promising agents, maybe one would make it to the point
3 of going through a clinical trial like this?

4 A. I know -- yes.

5 Q. We don't have to put a lot of effort into
6 finding 5,000 promising agents discovered in the
7 laboratory, entering non-clinical testing, five enter
8 phase I and one is approved?

9 A. It goes through phase III randomized pivotal
10 trial and gets approved.

11 Q. Does that mean you have proved that 4,999 don't
12 work?

13 A. I think some good drugs may be lost in the
14 process. I don't think we lost too many but those are
15 the numbers that we see. So it's a very small number
16 that make it all the way to approval.

17 Q. I just want to clarify. You got the end point
18 of what I was asking, which is some might be lost, but
19 is it a conclusion of the process that starts with
20 5,000 promising agents and ends up with one approval,
21 the process, the logical process that you're engaged
22 in, can you conclude from that process that the 4,999
23 have been proven not to be useful?

24 A. If they don't pass certain hurdles along the
25 process, they will be discarded. You would like to

1 better. That wasn't much, but it was better than the
2 current available therapy. In my mind six weeks of
3 improvement in my lifespan when I have to spend half of
4 it in the hospital getting treated is not such a great
5 breakthrough, so that is a disease that really needs
6 help but there was a drug that provided something
7 better than the standard at the day.

8 Q. Let me take a side issue and ask you about
9 Justice Ginsberg. Did you read anything about her
10 situation? This is a side issue completely but what is
11 your thoughts?

12 A. I can't comment. I don't know the extent of
13 her disease. They thought they caught it earlier but I
14 read it in The New York Times. She had a great
15 surgeon. I know him very well.

16 MR. J. TURNER: Just a side issue, I didn't
17 mean to take us off the record here, off the focus.

18 Q. In the time you have been involved with cancer
19 as a treating doctor and then doing the research you
20 described, are there any drugs that are used for cancer
21 therapy that are, quote, off label?

22 A. Depends what part of the world you're in.

23 Q. In the United States?

24 A. In the United States, yes.

25 Q. What is the story about that? How does that

1 discard them, recall, before you invest too many
2 patients, you don't want to waste resources today.
3 They're limited.

4 Q. Let me do a comparison and see -- I'm trying
5 to -- I don't know if it's a philosophical point or
6 logical point, but when you get done with your process,
7 5,000 promising agents, one of which went through the
8 whole process, you feel confident that you have
9 established something that is useful and meets the
10 criteria that we would like to see in the therapeutic
11 world?

12 A. Absolutely, yes, whether it's going to be
13 blockbuster breakthrough that really improves outcome,
14 not necessarily. There have been some drugs that have
15 been approved to treat diseases that are horrible. In
16 my mind pancreatic cancer is the worst cancer that
17 anyone can have. It's diagnosed late and there's not
18 effective curative therapy, but a drug that was
19 approved in the turn of the century to treat pancreatic
20 cancer was a breakthrough --

21 Q. Turn of which century, from --

22 A. 1990 --

23 Q. 1990 to 2000?

24 A. Yes. It improved survival compared to the
25 control arm by maybe six weeks, and quality of life was

1 work?

2 A. For a drug to be approved, it has to go through
3 that process that we just talked about. So that the
4 label is based upon the clinical trial that was done
5 for a certain disease type, certain cancer, certain
6 stage of the disease, a certain phase of its treatment.
7 Is it second line after somebody has had primary
8 therapy or is it first line. So that the label has --
9 these are the indications for its use.

10 Oncologists are studious people. They're
11 learning all the time and read the medical literature
12 and go to medical meetings and they hear a presentation
13 about that drug being used for not lung cancer but
14 pancreatic cancer. Although it's not been through the
15 pivotal trial to get approval for pancreatic cancer,
16 the aim of the study is to get there eventually. That
17 oncologist knows it may be helpful in his patient with
18 pancreatic cancer and doesn't have anything else and he
19 can write out a prescription.

20 Medicaid is going to approve off label drugs of
21 some drugs in phase II, early stage III.

22 Q. Are all the off label uses of drugs in phase
23 trials and new indication?

24 A. I don't think you can take something that no
25 one has ever looked at before and hope to use it in the

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1 patient but there should be some evidence, not pivotal
 2 trial, enough to get approval, that it is safe. In
 3 Europe you can't do that. If a drug isn't approved by
 4 the European National Health Authority, the doctors
 5 can't write a prescription and get it covered by the
 6 health agencies in that country unless they're
 7 financially well off and go get it somewhere else.
 8 So we have a lot of off label use but there has
 9 been some liberalization about that, depending on other
 10 studies, to support the use of the drug. Just last
 11 week Medicaid -- I always get mixed up.
 12 Q. Medicaid is old people over 65.
 13 A. Us old people over 65. There is a drug called
 14 Avastin, A-V-A-S-T-I-N, it's an antiangiogenic agent,
 15 A-N-G-I-O-G-E-N-I-C, and it's a monoclonal antibody
 16 and it goes after the factor that actually stimulates
 17 new blood vessel formation. It's approved for the use
 18 with chemotherapy in colorectal cancer and recently
 19 approved in non-small cell lung cancer and breast
 20 cancer but there is evidence to suggest it may be
 21 helpful in treating brain tumors and looks like that
 22 agency, Medicaid, is going to permit physicians to
 23 write prescriptions to use it with chemotherapy in
 24 brain tumors.
 25 Q. When you say "permit" --

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1 A. They're going to reimburse for it, that's
 2 right. But it's interesting, in the United States if
 3 you're on a clinical trial, a lot of the health care
 4 providers are obligated to cover the cost of clinical
 5 trials.
 6 Q. Aren't there other constraints by what they
 7 call experimental drugs?
 8 A. Some may be, but generally the understanding in
 9 many states is if a patient is enrolled in a clinical
 10 trial, and I believe clinical trials are good for
 11 patients because they get very, very careful care,
 12 followed very carefully, seen more frequently,
 13 responses are evaluated, safety issues are taken care
 14 of and get all the other supportive care that a cancer
 15 patient needs. Many carriers are actually covering the
 16 cost of clinical trial. They don't provide the drugs.
 17 The drug company is going to provide the drug, but what
 18 the health insurance carrier will cover is a lot of the
 19 laboratory expenses, the clinic expenses and even the
 20 medical imaging expenses which would generally be
 21 standard. Clinical research isn't hard to do in the
 22 country. It's getting patients to be willing to
 23 participate.
 24 Q. Do you know how much off label use there is?
 25 A. Varies from drug to drug. I don't have a

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1 number off the top of my head.
 2 Q. Is there off label use by people writing
 3 prescriptions for things that they will not have
 4 reimbursement for from, say, Medicaid or Medicare?
 5 A. Probably not.
 6 Q. Okay. I wanted to ask you, you gave an
 7 indication of materials that you reviewed getting
 8 prepared for this process.
 9 A. Yes.
 10 Q. Could you just go through that again very
 11 quickly?
 12 A. Again, this is not in specific order but --
 13 Q. You don't have to do it extensively because we
 14 have it in writing, but just a quick rough summary.
 15 A. I reviewed the literature citations that were
 16 provided by Daniel Chapter One. I have them listed all
 17 here.
 18 I reviewed the deposition testimony of James
 19 and Tricia.
 20 I reviewed the transcripts from two of their
 21 Healthwatch Radio Programs that were done in July of
 22 this year.
 23 I reviewed the testimonials of the 30 patients,
 24 some who had cancer, some who didn't. These were
 25 testimonials submitted by patients or sometimes

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1 relatives or sometimes friends of the patients who had
 2 used the Daniel Chapter One products.
 3 I mentioned the complaint. I reviewed their
 4 bioguide, Biomolecular Guide for Daniel Chapter One
 5 listing all of the different products that they have in
 6 their company.
 7 I reviewed recently -- I don't have it in my
 8 report because I think it came in after I submitted it.
 9 It was an extensive listing of all the different
 10 diseases, not just cancer, but every disease imaginable
 11 or condition for which an individual could take one or
 12 several of Daniel Chapter One.
 13 Q. Do you know what that document was?
 14 A. Something for physicians, simple guide for
 15 doctors, so it was really geared for physicians to look
 16 this up and say, okay, I have a patient with cancer,
 17 which is a lot of different disorders, but this one had
 18 cancer as one single entity and listed a number of
 19 different products.
 20 Q. Who prepared this document?
 21 A. Daniel Chapter One.
 22 Q. Is that something you can provide to us?
 23 MR. PAYNTER: I think they were supposed to
 24 send it to you. So I have to check with David to see
 25 whether they did.

1 MR. J. TURNER: I don't recognize it.
2 MR. PAYNTER: It would have been in the last
3 day or so.

4 MR. J. TURNER: I don't recognize that, so --
5 A. I did review yesterday, because I just got them
6 yesterday, the expert reports from a number of the
7 experts for Daniel Chapter One. Then I did my own
8 literature search, and sources of that are in my
9 report. I have specific references supporting the four
10 different sections of my report for Bio*Shark, GDU,
11 BioMixx and 7 Herb Formula or in the appendix with the
12 specific references supporting those segments of my
13 report.

14 Then I did extensive searches of Google and
15 Memorial Sloan Kettering, Dana Farber, I used Stanford
16 HighWire, PubMed, Clinical Trials.gov gives you all the
17 clinical trials ongoing by different disease entities.

18 The journals I read that I get, subscribe to
19 them that are listed here. That includes Journal of
20 Clinical Oncology, New England Journal of Medicine,
21 British Journal of Hematology. I was on the editorial
22 board of that one and another, Supportive Care in
23 Oncology, which covers a lot of the alternative and
24 complimentary medicines. A very helpful book that was
25 written by Barry Cassileth and Lucarelli at Memorial

1 Sloan Kettering, "Herb Drug Interactions in Oncology."
2 It lists a lot of the different individual compounds in
3 some of the DCO, Daniel Chapter One, products, just
4 from some literature, if it's supported, pre-clinical,
5 non-clinical studies, if any were done.

6 Then my own experience, because I've done a lot
7 of work in the field of alternative medicine when I was
8 at Cancer Treatment Centers of America, and believe it
9 or not, we still see protocols and requests for
10 proposals coming from the pharmaceutical industry or
11 the neutropharaceuticals industry asking us to help
12 them design and conduct clinical trials looking at
13 alternative therapies in the treatment of cancer. So
14 we're doing that today.

15 Q. Can you give me an indication of --

16 A. I can't give you the specific names. I can
17 give you a general overview. This is a product that
18 has come from a mushroom, mushroom extract.

19 Q. Is that the one you mentioned?

20 A. No. I did that study at Cancer Treatment
21 Centers of America. This is another one that came from
22 a company. Confidentiality doesn't permit me to say
23 anymore, help us with phase I, II and beyond, looking
24 at product with conventional chemotherapy to see
25 whether patients might have tolerated treatment better,

1 less side effects and maybe have a better response to
2 disease progression.

3 So it was going to be phase I where you find
4 out what the best dose might be and look at
5 pharmacokinetics, K-I-N-E-T-I-C-S, where we see whether
6 there is any interaction between their product and the
7 conventional chemotherapy that might either have an
8 effect in keeping concentrations too high or lower in
9 their concentrations so they don't work.

10 Also seeing whether it might increase toxicity
11 of the chemotherapy or lower its efficacy and find out
12 what the best dose might be to move into a phase II
13 trial, which in this case can be randomized trial.
14 Patients would be randomized, in this case double blind
15 placebo controlled trial. You can find a liquid that
16 looks and tastes, buy it and randomized for
17 conventional chemotherapy for their disease with their
18 product or a placebo and see if you can meet the end
19 points and design the study so you have enough patients
20 in each arm to meet what you set up as a null,
21 N-U-L-L, hypothesis and say there is no difference
22 between response rates in patients getting mushroom
23 extract X or placebo. And you're basically going to
24 disprove the null hypothesis by showing there is a
25 statistical difference between the two that is not

1 based on chance alone. Then you've shown what we would
2 call reliable and competent evidence that this agent
3 actually increases the response rate in patients with
4 that particular disease.

5 (A recess was taken.)

6 Q. Couple of questions before we go on to the next
7 section, part two of the report. You've described a
8 fairly elaborate system for reviewing processing
9 agents. Is that because they tend to be toxic?

10 A. That is not the only reason. Safety is an
11 important part of the evaluation of a new drug, but the
12 efficacy is also important as well as the pharmacology,
13 pharmacokinetics.

14 Q. What is the pharmacokinetics?

15 A. Pharmacokinetics means how is the drug absorbed,
16 how is it distributed in the body, how and where is it
17 metabolized, where or how is it excreted, what's the
18 maximum level you can get in the blood, if you give it
19 by mouth, does it get absorbed. So what is its
20 bioavailability. If you give a compound by mouth and
21 it gets into the stomach and the stomach acids break it
22 down and activate it, you can't measure anything in the
23 blood. It may not be absorbed. There are certain
24 things that can't be absorbed, blocked.

25 Q. Is there a significant number of drugs that go

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1 through phase I, II and III studies, trials, that do
 2 not have a toxic component?
 3 MR. PAYNTER: I just object. In general or
 4 are we talking about oncology? Because you said --
 5 MR. J. TURNER: Make it oncology.
 6 A. Every drug has some kind of, you call it toxic,
 7 I would say some ad effect or adverse effect, yes.
 8 Q. Go ahead.
 9 A. It's okay.
 10 Q. If I didn't get the questions we talked about
 11 in the break, I'll get them at the end, but now we're
 12 going to go to that part of the report that's part two,
 13 "Scope of Work."
 14 You indicate that there are I think eight
 15 statements that you wrote here as you're looking for
 16 evidence to substantiate the following claims. Did you
 17 write "Bio*Shark inhibits tumor growth" as one of the
 18 claims?
 19 MR. PAYNTER: Objection.
 20 A. I wrote --
 21 MR. PAYNTER: What do you mean, did he
 22 physically write it or did he --
 23 A. What's in here I wrote.
 24 Q. What I'm asking you is, where did you get those
 25 words?

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1 A. They came from a section in the complaint. I
 2 don't recall the exact number.
 3 Q. Is that true for all of these?
 4 A. This is I think verbatim from the complaint.
 5 Q. From the complaint, okay. Actually, one of the
 6 questions I meant to ask you before we got to this, but
 7 that's a good beginning of that, I wanted to ask you if
 8 you had in your review of materials, had you reviewed
 9 any of the German monographs on herbs?
 10 A. Not the monographs, no.
 11 Q. Are you familiar with the monographs?
 12 A. I'm aware of them, I heard about them, but I
 13 did not read them.
 14 Q. Did you look at the United States Pharmacopeia
 15 on Herbs?
 16 A. Again, I'm aware of that but I did not read it.
 17 Q. How about the British Pharmacopeia?
 18 A. Did not read it.
 19 Q. Did you review the Complementary and
 20 Alternative Physician's Guide?
 21 A. Can you expand that? Which one?
 22 Q. It's published by Springhouse Publishing and
 23 it's the Guide to Complementary Physician Practice?
 24 A. I did not read that.
 25 Q. Did you review any material at all by Dr. James

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1 Duke?
 2 A. The only thing I read of Dr. Duke was his
 3 report. I did not read any of his listed publications.
 4 Q. You didn't look at the online database that he
 5 maintains at the U.S. Department of Agriculture on
 6 herbs?
 7 A. I did not.
 8 Q. I was going to ask, did you review anything
 9 from the American Botanical Council?
 10 A. No, I did not.
 11 Q. You indicated that you had reviewed -- I gather
 12 this list in your report is things that you reviewed.
 13 The part that says materials that I reviewed has a list
 14 of documents that apparently are those that were
 15 provided by -- given to you as having come from Daniel
 16 Chapter One. It's a list. Do you know what I'm
 17 speaking of here?
 18 A. No.
 19 Q. "I have also reviewed the following material
 20 provided to me by the FTC." Let me ask you about this.
 21 What did you learn from the transcripts of the radio
 22 programs?
 23 A. I learned that people with cancer called in,
 24 gave a brief capsule of their diagnosis or what they
 25 were advised to do and it might be surgery or might be

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1 radiation therapy or might be chemo or combinations,
 2 and they were given advice about what to do about their
 3 disease. Don't go through cancer therapy. Don't get
 4 radiation, chemotherapy is bad for you. Chemotherapy
 5 has never cured anybody. My relative had that and she
 6 died from it. There was advice being given to cancer
 7 patients about what they should do about the treatment
 8 of their disease. That was one thing I learned.
 9 Q. Let me ask, do we have transcripts of those?
 10 MR. PAYNTER: They would have all been
 11 produced.
 12 MR. J. TURNER: The transcripts themselves.
 13 A. That's what I learned. The rest was some other
 14 thing, discussing the products, but that is the primary
 15 bottom line thing that I learned from those radio
 16 programs.
 17 Q. The next thing was testimonials submitted by 30
 18 patients. How did you receive those 30 patients'
 19 testimonials?
 20 A. I think each of the patients had a one, two --
 21 one-page narrative of who they were, what their cancer
 22 was and what they did to treat it, what products they
 23 took and how they were benefited by it.
 24 Q. This was given to you by the FTC?
 25 A. Yes. Some of those testimonials appear in

1 other DCO materials on their web site or other of their
 2 documents.
 3 Q. Then continuing down it says articles -- can
 4 you find the place in your report -- you got that?
 5 A. Yes.
 6 Q. "Articles for research study of
 7 complimentary/alternative proprietary products in
 8 support of respondent's claim, (appendix III)."
 9 A. Yes.
 10 Q. What does it mean by alternative proprietary
 11 products?
 12 A. Well, I think that title came from DCO, but I
 13 don't think I wrote it that way. I think that's how
 14 they listed it in their responses.
 15 Q. Okay.
 16 A. So I don't know what they mean by
 17 complimentary/alternative proprietary products.
 18 Q. You have other cited articles and those are
 19 cited by whom?
 20 A. These are literature provided by DCO.
 21 Q. Then I wanted to ask you about some of those.
 22 That is the list I was looking for. Did you look at
 23 Dr. Nieper's "Revolution in Technology Medicine and
 24 Society"?
 25 A. I looked at all of these things here. I had a

1 beginning clinical trials to suggest that curcumin,
 2 which is from tumeric, may be -- may warrant additional
 3 studies to see if it can prevent particularly
 4 colorectal cancer. There have been a number of
 5 peer-reviewed articles suggesting that that particular
 6 compound, curcumin, is worthy of further investigation
 7 and I go into that in my report.
 8 Q. We're going to talk about that. Then there is
 9 one which is Foster, S. Echinacea, "Helping to Rebuild
 10 Your Immune System."
 11 A. No literature support -- this was just an
 12 opinion article with not very much supported data for
 13 what he is trying to say.
 14 Q. Do you have a sense of the immune's
 15 relationship to all of this dynamic that we're
 16 discussing?
 17 A. You made it sound so general, and it's much
 18 more specific.
 19 Q. Make it specific.
 20 A. The immune is important in fighting cancer, or
 21 the immune is suppressed in cancer patients, so if we
 22 beef up the immune, we can destroy the tumor, it's more
 23 complex than that.
 24 Q. These are not cancer people. These are just
 25 the whole world. If you beef up your immune, you'll be

1 stack of stuff.
 2 Q. What was your take away from the Nieper
 3 Revolution?
 4 A. I don't recall while I'm sitting here right
 5 now.
 6 Q. That's fine.
 7 A. I just don't recall.
 8 Q. On the Majeed M. Badmaev and Murray F. Tumeric
 9 and the Healing Curcuminoids, what was your take on
 10 that or take away from that?
 11 A. I'm going to make a general statement first and
 12 that is throughout this whole process. I relied on
 13 peer-reviewed articles that went through the normal
 14 process of review by experts and peers in the field.
 15 That's how we publish things in science. If an article
 16 contained reference to peer-reviewed articles, that was
 17 empty to me. If it was subjective review of the use of
 18 a product somewhere, like many of the pharmacopeias
 19 have without peer review, supporting data, to me the
 20 evidence was not as strong as somebody writing
 21 subjectively about their own opinions. That wasn't
 22 what I was relying upon.
 23 If I recall the Tumeric and Healing
 24 Curcuminoids, I will agree that there had been a number
 25 of very interesting non-clinical studies and some

1 healthier?
 2 A. As a general statement?
 3 Q. Yes.
 4 A. What if it's normal to begin with. Do you have
 5 to beef it up further to be healthier?
 6 Q. That is my question.
 7 A. I don't know.
 8 Q. Your argument would be if it's below normal,
 9 yes, but if it's normal we don't want to necessarily do
 10 that?
 11 A. Do you know what happens if you over beef up?
 12 You get auto immune, lupus, and maybe neurological
 13 disorders, so beefing it up, if it doesn't need to be
 14 beefed up, why do it?
 15 Let's beef up another system. Let's beef up
 16 the blood system. Hemoglobin in our body carries
 17 oxygen from the lungs to the tissues and then it
 18 carries the carbon dioxide back to the lungs and we
 19 breath it out. Normal hemoglobin for you is 14, 15, 14
 20 to 15 grams of hemoglobin per hundred MLs of your
 21 blood. Gee, let me make it up to 18, you'll be better
 22 because it's beefing it up. And you know what is going
 23 to happen, you'll clot something in your brain and have
 24 bad effects, so more isn't better. If it's too low,
 25 that is not good. Beefing it up may not be beneficial.

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1 Q. You're saying just like the blood system, that
 2 would be true of the immune?
 3 A. In many respects, yes. If I have normal immune
 4 I don't need to have it beefed up unless I have
 5 deficiencies. There are some diseases where we talk
 6 about gamma globulins. They are the proteins that help
 7 the body fight viral infections, fungal infections,
 8 maybe important in identifying foreign substances in
 9 our body. There are diseases where you make too many
 10 gammaglobulin because the cells are abnormal and it's a
 11 disease called multiple myeloma.
 12 Q. Is cancer a disease?
 13 A. Of course.
 14 Q. And when you're at ten to the four, do you have
 15 cancer or not?
 16 A. You do not have cancer.
 17 Q. What do you have?
 18 A. I don't know what you have because I'm not
 19 sure -- ten to the four may remain that way for the
 20 next 40 years.
 21 Q. And --
 22 A. Cancer is a diagnosis based on physical
 23 findings, laboratory findings, medical imaging
 24 findings. It's not lurking where it's not detectible.
 25 Q. So people who have -- people who show up with

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1 cancer that is ten to the 11 I guess you said --
 2 A. That was one particular type. Let's not
 3 generalize. Cancer is one disease, we can't say that.
 4 We have to separate things.
 5 Q. Here is what I'm trying to understand. At a
 6 given moment you are able to diagnosis something as the
 7 disease cancer?
 8 A. When it reaches a certain size, when there is a
 9 certain number of cells in a mass that is detectible by
 10 some medical imaging, CT scan, MRI, a bone marrow test,
 11 biopsy.
 12 Q. Before that you're healthy?
 13 A. Yes.
 14 Q. So a given day you're at ten to the five and
 15 the next day you're something greater than that until
 16 it manifests yourself, you're healthy at that point?
 17 A. You can't say you're ten to the fourth one day
 18 and the next day you're ten to the fifth because
 19 different tumors and different malignancies grow at a
 20 different rate. There is also a rate where tumor cells
 21 may die.
 22 Going back to your example of ten to the fourth
 23 or third, there may be a balance. There are cells that
 24 are growing and multiplying -- let me answer the
 25 question. There are cells multiplying and dividing and

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1 one cell becomes two. That is the growth rate. But at
 2 the same time there is an innate cell death rate. So
 3 some cells are dying. They go into what we call a
 4 programmed cell death.
 5 So cells are not constantly multiplying and
 6 dividing. There are some cells dying, multiplying and
 7 it may be balanced and it may remain ten to the three
 8 forever if that is the balancing effect.
 9 Q. What you're saying is in the whole universe of
 10 people that get ten to the three, some of them may be
 11 balanced?
 12 A. That's right. They may never have diagnosable
 13 cancer.
 14 Q. In the whole universe of people who get to the
 15 ten to the 11, is there anyone who never went to ten to
 16 the third?
 17 A. Of course. You don't just suddenly come up
 18 with --
 19 Q. You can't do that. So the universe of people
 20 who end up with tumors are people who started out
 21 probably somewhere below that and evolved to that?
 22 A. Yes, that's correct. What we're trying to do
 23 now is come up with molecular biological techniques to
 24 see if we can identify certain known abnormalities in
 25 cells that would go along with the development of a

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1 malignancy.
 2 Let me give you an example. There is a
 3 condition called chronic amyloid leukemia. There is an
 4 over production of white blood cells. It can go on for
 5 three, four, five years. Until recently there is a
 6 specific treatment to go after the molecular,
 7 biological defect in chronic amyloid leukemia, an
 8 abnormality in the chromosome where a piece of one
 9 chromosome hooks up to a piece of another chromosome,
 10 because they develop -- they dissolved it in
 11 Philadelphia. It's called the Philadelphia chromosome.
 12 People who have chronic amyloid leukemia, many of them,
 13 not all, have this Philadelphia chromosome.
 14 This new drug goes after the place where the
 15 two chromosome pieces are connected together and gets
 16 rid of the cells. And patients can be put into a
 17 remission where the white blood cell goes down to
 18 normal. You don't see the Philadelphia chromosome any
 19 longer and the next material level of making sure they
 20 don't have disease is you can't see any of the
 21 combination of the chromosome. There is a very fancy
 22 technique we can use for that. There is a limit of
 23 detection we can get down for that test, maybe ten to
 24 the minus one. So we can get down to very few cells.
 25 I guess you could screen people to see whether

1 they were expressing this chromosomal abnormality.
2 It's unlikely today in science if we were to detect a
3 very few of these Philadelphia chromosome positive
4 cells that were harboring this molecular fusion,
5 F-U-S-I-O-N, that we would begin treatment for those.

6 Q. Say that again?

7 A. We would not begin treatment for a patient like
8 that. Even though -- that might be the hallmark of
9 chronic amyloid leukemia. We don't usually treat
10 patients until they've got clinical --

11 Q. Why is that?

12 A. We're not sure whether it might be more harm
13 than good. There are patients -- prostate cancer.
14 Prostate cancer, if you live long enough and you're
15 male, you will develop it probably. So many men die,
16 at autopsy they have prostate cancer and never knew it.
17 There are many men who have low grade prostate cancer,
18 not aggressive, and they may not need any treatment for
19 it at all and live a healthy, normal life without
20 needing surgery, radiation therapy and certainly not
21 chemotherapy. There are diseases that are very low
22 aggressiveness and you can live with them for a long
23 period of time.

24 We have to look at cancer sometimes as a
25 chronic disease that our bodies may have to learn to

1 manifestations of recurrent disease.

2 Q. Do you know if diet has an impact on that
3 question?

4 A. Diet is important for any cancer patient.

5 Q. How do you interface diet with a cancer patient
6 in a situation that you just described?

7 A. Which one?

8 Q. The one --

9 A. Philadelphia chromosome one or breast cancer
10 patient?

11 Q. You just described one where there was a small
12 amount of circulating cancer cells.

13 A. For that situation, except for general
14 principals of restriction of fatty intake and vegetable
15 and fruits and making sure you get nutritious foods,
16 I'm not sure of any specific nutritional evidence that
17 something else would be better.

18 Q. For that situation you're not sure there is
19 anything. Are there any situations that are analogous
20 to that where you would have some idea about nutrition?

21 A. In a patient who already has been diagnosed
22 with cancer?

23 Q. We can start with that.

24 A. I wouldn't answer it any differently than I did
25 before.

1 live with without necessarily eradicating it. I prefer
2 to eradicate acute lymphoblastic leukemia in a child.
3 I want them to get rid of it but we have very sensitive
4 techniques now to measure residual tumor cells. For
5 example, a woman with metastatic breast cancer could
6 get treated with surgery -- with chemotherapy and I can
7 take a small amount, little more than a teaspoon full,
8 and I can identify cancer cells circulating in her
9 blood stream. And if there are a certain number of
10 those, not very many in that teaspoon and a half of
11 blood, if there are five or more circulating tumor
12 cells, I know that that woman is at a greater risk of
13 developing a reoccurrence of her disease even though
14 she doesn't have one now.

15 Q. So would you take --

16 A. What I would do, and that's what is being done,
17 let's see whether treatment now is better than waiting
18 until she really has evidence of metastatic disease.
19 It's an unknown question. You pose a scientific
20 question, is it more effective to treat somebody at
21 this first evidence, microscopic evidence of
22 reoccurrence or wait until the disease recurs. We
23 don't know the answer to that. You may be putting
24 people at harm if you treat them and may not be any
25 difference if you wait until they have the first

1 Q. How about somebody who you detected this small
2 amount of circulating cancer cells who has not been
3 diagnosed ever before?

4 A. I don't know the answer. I don't know whether
5 dietary manipulation and giving a patient Tracrium is
6 going -- whether giving them heavy metals of some kind
7 or elements of some kind is going to prevent them from
8 developing breast cancer. I don't know the answer.

9 Q. We've used some words that I just wanted to get
10 your take on, what they mean when you use them. The
11 first one is "drug." What do you mean by "drug"?

12 A. A drug is generally a chemical or
13 pharmaceutical that can be either synthesized or can be
14 a natural product that is used in a specific dose by a
15 specific route of administration to treat a medical
16 condition, in some cases prevent certain medical
17 conditions, and is given for a finite period of time in
18 a specific dose and dose schedule.

19 Q. Then another word that we've used a lot is
20 "disease." How would you describe the word "disease"?

21 A. Well, we have a state of normalcy and we have a
22 state of medical abnormalcy. I would consider a
23 disease abnormal state of health.

24 Q. In the progression from non-expressed cancer to
25

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1 expressed cancer, and the example we have been using,
 2 starting with ten to the first --
 3 A. One cell, ten cells?
 4 Q. Ten cells, ten to the 11th, is there a place in
 5 that progression that disease begins or manifests and
 6 how would you describe that? What would that place be?
 7 A. In terms of number or just in terms of clinical
 8 manifestation?
 9 Q. Clinical manifestation.
 10 A. Leukemia as an example. You have to understand
 11 what the disease is all about. And it's the
 12 advantageous growth and multiplication of leukemia
 13 cells in the bone marrow, that's where they're made,
 14 where the growth of the leukemia cells actually is much
 15 greater and faster and crowds out the normal bone
 16 marrow cells that produce red blood cells or white
 17 blood cells or platelets. What happens is that the
 18 bone marrow becomes filled up with leukemia cells and
 19 some of those may spill out into the blood stream.
 20 In the process of crowding out the bone marrow,
 21 because it's basically taking over because of the
 22 advantages of the leukemia cell and multiplying and
 23 dividing, if it's a rapid process, you might get from
 24 the replacement of the normal bone marrow by leukemia
 25 cells, you might get bone pain, back pain. You might

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1 get joint pain.
 2 So one of the earliest manifestations in a
 3 child, they complain of aches and pains. In three or
 4 four-year olds who are busy all the time, it's not
 5 considered to be anything. Sometimes if it's a rapidly
 6 growing process, the lymph nodes get filled up with
 7 leukemia cells also. So a child can have enlarged
 8 lymph glands in the neck, under the arm and it's
 9 considered to be a viral infection unless somebody does
 10 a blood count. If they do a blood count, they can see
 11 a number of different things, depending on how rapidly
 12 the disease is multiplying and dividing and how much
 13 cell death there is. It's not one process.
 14 So some children, because their marrows have
 15 been over taken by the leukemia cells and are not
 16 making red blood cells, they become anemic and the
 17 child looks pale. It may not be noticed if it's
 18 wintertime. Kids look pale in wintertime unless they
 19 live in Florida or California. They may have infection
 20 because they don't have normal white blood cells to
 21 fight the infection. They may have fever. If they're
 22 not making blood platelets, they may bruise easily,
 23 more so than they usually do.
 24 Hematologic manifestations are related to the
 25 decreased production of normal blood cells. The fever

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1 may be related to the disease process itself and some
 2 of the biochemicals that the body produces to
 3 counteract the leukemia, which can cause fever. And
 4 the bone pain and joint pain is filling up the bone
 5 marrow with tumor cells.
 6 Some kids may present with severe headache and
 7 may have leukemia cells in the brain or spinal fluid.
 8 Others may have leukemia cells in the liver or spleen,
 9 which get enlarged. I've seen patients who have
 10 leukemia cells in their intestinal tract and it
 11 perforated and they presented with what looked like
 12 appendicitis but was really leukemia. Those are the
 13 early clinical manifestations of the disease. If you
 14 suspect it, you do a blood test and you can often see
 15 leukemia cells in the blood smear and you can see
 16 changes in the platelet count or the hemoglobin level.
 17 Q. When you reach that clinical state, what is the
 18 proper course of action?
 19 A. Once you established the diagnosis, you then do
 20 other studies to help you with prognosis. We look in
 21 the chromosomes, not the one I was talking about
 22 before, that is chronic, but in acute leukemia we look
 23 at chromosomes in good laboratories. In Memorial Sloan
 24 Kettering they look for some of these molecular
 25 abnormalities that are part of the molecular genetics

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1 of the disease. We look at the biochemical picture of
 2 the patient because we have to support them very
 3 carefully when we start their treatment to make sure
 4 the kidneys are going to function normally.
 5 The next step, once we established the
 6 diagnosis and know where it is, we want to make sure
 7 it's not in the central nervous system, patients are
 8 started on chemotherapy.
 9 Q. Drugs?
 10 A. Yes.
 11 Q. Do all those drugs have a toxic side effect?
 12 A. I said earlier every drug has a toxic side
 13 effect. Herbals have a toxic side effect.
 14 Q. We talked about drugs, disease. What is a
 15 cure?
 16 A. It depends on the disease. If we're talking
 17 about acute lymphoblastic leukemia, generally if a
 18 patient has gone four or five years from the time that
 19 therapy has been completed, and they've never had
 20 disease reoccurrence, I would say 95 percent plus of
 21 those patients are going to be cured.
 22 Q. Do you have statistics on the life of these --
 23 this covert of people, that is the group that has gone
 24 say five years, do you have statistics on the rest of
 25 their lifespan?

1 A. I can give you statistics or I can give you my
 2 own personal experience. What would you like?
 3 Q. Both.
 4 A. Children who have a malignancy of any kind, and
 5 leukemia is one kind, who are treated with
 6 chemotherapeutic agents and some received radiation
 7 therapy, a small proportion, a very small proportion,
 8 few percent, may be at risk of developing a second
 9 malignant neoplasm at a later date.
 10 When we treated children with acute
 11 lymphoblastic leukemia, we knew that leukemia cells
 12 were either in the central nervous system or can get in
 13 there. And in the early days, all of the children not
 14 only got treated with chemotherapy, but also radiation
 15 therapy to their brain and the spinal canal to prevent
 16 central nervous system leukemia.
 17 In a certain group of patients began a very
 18 small percentage, under three or four percent, in a
 19 particular age group under ten years of age, some of
 20 those patients went on to develop brain tumors related
 21 to either some genetic pre-disposition and/or the
 22 results of or the effects of therapy.
 23 Now we've learned that certain patients don't
 24 need radiation therapy. We don't use it and they get
 25 treated with chemotherapy that's given directly into

1 when she was a kid and doesn't want to run that risk.
 2 So it's mostly the guys who are afraid of marrying a
 3 young lady who has leukemia so the marriage rate is
 4 lower.
 5 Now that we're not using radiation therapy,
 6 we're not seeing the neuropsychological cognitive
 7 defects, but I think those are the major. There are
 8 some effects on organs of the body. If chemotherapy
 9 might damage the liver, they usually get over it.
 10 Central nervous effects are not as severe as they were
 11 before.
 12 The other effects of treatment might be related
 13 to some of the specific drugs that were used that have
 14 heart toxicity or liver toxicity where there may be
 15 some effects.
 16 Q. How does this compare to adults who are treated
 17 for cancer and reach a five-year survival rate?
 18 A. With adults, five-year survival is generally
 19 interpreted as a good sign. We know in certain
 20 cancers, breast cancer, there may be late recurrences
 21 so five-year survival doesn't necessarily mean cure,
 22 although the survival curves tend to flatten out at
 23 that period of time.
 24 Adult patients don't tolerate chemotherapy as
 25 well as children do for a number of reasons. It's the

1 the spinal fluid. We also learn that chemotherapy
 2 might have an effect on the growth of a child because
 3 it effected the pituitary gland. So the children had
 4 lower growth because they had less growth hormone and
 5 they often were obese, and the third adverse effect of
 6 radiation therapy was that some of the children,
 7 particularly the young ones, had a neuropsychological
 8 dysfunction, learning disabilities from the effects of
 9 radiation therapy.
 10 It was through clinical trials and primarily
 11 that we now do not use radiation therapy for most
 12 patients with acute lymphoblastic leukemia, so we're
 13 obviating the effects on growth, the effects on obesity
 14 and the neuropsychological defects. Otherwise, I think
 15 these children live, and the data would support this,
 16 they live good lives. They have trouble getting jobs,
 17 interesting.
 18 Q. Why do they have trouble getting jobs?
 19 A. Insurance companies don't want to give them
 20 coverage even though they had leukemia and they're
 21 cured. I think their marriage rate is lower. I have
 22 seen that from my own patients who are wonderful
 23 people, cured of their leukemia, they're bright,
 24 beautiful, vivacious and every time they meet somebody,
 25 the guy gets scared because he heard she had leukemia

1 nature of their tumors that are not responsive, as
 2 responsive to chemotherapy as many of the pediatric
 3 tumors are. The adult patients have a lot of other
 4 lifestyle things that effect organ function, the
 5 smoker, drinker, the both, patients who are obese, who
 6 have hypertension, they may have diabetes and a lot of
 7 other comorbid medical conditions that make treating
 8 their disease more problematic.
 9 Adult patients maybe are not as tolerant of
 10 some of the side effects of chemotherapy, like nausea
 11 and vomiting, even though we have medicines now to
 12 decrease that. I think doctors will decrease or delay
 13 therapy in an adult patient, particularly if the adult
 14 patient complains about some of the side effects. We
 15 don't do that as much in pediatric oncology. So kids
 16 get more therapy. They may be tougher soldiers and may
 17 be one of the reasons they do better. Really
 18 interesting stuff. I need to talk about it because you
 19 asked about adults and children.
 20 Q. Go ahead.
 21 A. We'll take acute lymphoblastic leukemia. If
 22 that child is treated by a pediatric oncologist with a
 23 reasonable protocol, the results will be much better if
 24 the pediatric oncologist is treating, let's say, a
 25 16-year old. If that 16-year old happens to go to one

1 of my medical oncology colleagues using the same
2 protocol, the results are better with the pediatric
3 oncologist treating that 16-year old than the medical
4 oncologist because they're not as aggressive, chicken
5 out, I don't know what it is, being published and it's
6 really interesting.

7 So you have to understand the disease, you have
8 to understand the patients and what's at stake and why
9 it's so important to continue therapy. We have
10 supportive care for a lot of the side effects. You
11 can't say chemotherapy is terrible, everyone is going
12 to die, all these terrible things happen. We can treat
13 the anemia, low white blood cell counts, very effective
14 to treat serious infections, we have antibiotics -- I
15 don't mean stimulating their immune system to treat the
16 fungal infection. I want to get rid of the fungus and
17 need antifungal agents to do it. I can use medicines
18 to stop the nausea and vomiting. I can tell when
19 they're malnourished and put all those things into
20 place to treat them.

21 It's the whole patient. The whole patient in
22 cancer isn't let's just go after the body and forget
23 all the other stuff that kills them, that is not me
24 speaking, and I read their report. It's treat the
25 whole patient and understand all these different organ

1 systems and parts of the body are important. Don't
2 neglect any of them. And I think that's what we do in
3 oncology.

4 Q. Okay.
5 A. It's a big team caring for cancer patients
6 today, not just the oncologist injecting
7 chemotherapeutic agents in a patient.

8 Q. We talked earlier about early detection.
9 A. Yes.

10 Q. Are there tumors that go away by themselves?

11 A. Rarely there can be spontaneous remissions,
12 spontaneous disappearances of tumors. I've seen that
13 happen in tumors of the sympathetic nervous system
14 where a patient starts off with what appears to be a
15 malignancy and the patient's tumor goes from a
16 malignant tumor to a benign tumor and can be removed
17 surgically. We're looking at new drugs that actually
18 help that process of turning tumors that are mature to
19 go from a malignant state to a benign state.

20 Q. Say that again, I'm sorry.

21 A. We have drugs now that are designed to help a
22 tumor go from a malignant state to a more benign state,
23 because of maturation of the tumor, we call it
24 differentiation.

25 Q. Do you think of the products that you are

1 analyzing for Daniel Chapter One as drugs?

2 A. Again, any class of agent, I don't care what
3 you call it, any class of agent that's designed to
4 treat a disease, its basic disease or prevent a disease
5 is medicine, a drug. You can't separate conventional
6 medicine from alternative medicine if the aim is to
7 treat cancer. But there are different classes of
8 drugs, many different classes of drugs that fall into
9 what they're made of, what their chemical composition
10 is, what their target might be in the body.

11 Q. Do you have a way of thinking about classifying
12 the Daniel Chapter One products in one of those
13 category of drugs? I'd like to hear the answer?

14 A. Let's take Bio*Shark, B-I-O.

15 Q. For the record, we're going to go over each of
16 those in more detail.

17 A. Let's take Bio*Shark. From the work that was
18 done by the Harvard scientist back in the '80s, they
19 isolated from crude shark cartilage a peptide, protein.
20 This was highly purified. They started off with grams,
21 pounds of shark cartilage and came up with a few grams
22 of peptide. When they put it into a test tube or petri
23 dish with tumor cells or looked at new blood vessel
24 formation, they saw that this peptide from shark
25 cartilage actually prevented new blood vessel

1 formation. That's antiangiogenesis. One mechanism of
2 action of a drug would be antiangiogenesis active. I
3 think the shark cartilage is what that agent is
4 supposed to be doing.

5 Q. Okay. Do you think DCO, Daniel Chapter One,
6 thinks of these as drugs?

7 A. I don't know.

8 MR. PAYNTER: Objection. Objection. No
9 foundation. Objection.

10 Q. Have you read their materials?

11 A. Yes, I have.

12 Q. Based on your reading of their materials do you
13 believe they're thinking of these as drugs?

14 A. If they propose that their drugs can replace
15 conventional therapy, then yes, it's a drug.

16 A broader term would be anticancer agent. Some
17 of the things we use are monoclonal antibodies that
18 are a little bit different than a drug, but a drug has
19 a mechanism of action, excreted, metabolized in a
20 certain way so anything like that that's chemical or
21 structural formula that's used to go after a cancer
22 cell, is an anticancer agent.

23 Q. Is that true of a food as well?

24 A. What kind of food?

25 Q. Broccoli?

1 A. I don't think it has specific anticancer
 2 activities. It may provide nutrients that are
 3 important for the body and in certain circumstances may
 4 seem to have in a test tube some anticancer activity,
 5 green tea may. Other things we eat may.
 6 Q. Green tea would be a food in the way we're
 7 talking about now or a drug?
 8 A. If you're saying take these things because
 9 you'll feel better, they're good for you, they can't
 10 provide specific therapy for your cancer because it's
 11 not been proven, there is no competent or reliable
 12 evidence that these things work in treating human
 13 cancer. If they do no harm and may have some
 14 beneficial effects because they contain nutrients of
 15 some kind, I have no objection to that. I want to make
 16 sure my patients are getting good nutritional diets and
 17 getting enough calories and all the other things they
 18 need to be as healthy as possible. But I wouldn't ever
 19 substitute broccoli for Avastin and cisplatinum to
 20 treat their colon cancer.
 21 Q. Do you believe that is what Daniel Chapter
 22 One --
 23 A. I think they said it. I read it in their radio
 24 reports. If you read into the next layer beyond the
 25 label of their products and look at the pages in their

1 you were preparing your report?
 2 A. I may have.
 3 Q. If you may have seen it, how would you have
 4 treated it as far as your report goes?
 5 A. Well, there are other things in their web site
 6 and documents you can download on their web site that
 7 contradicts that and also things that they've said.
 8 Tricia gave --
 9 MR. PAYNTER: There is no question.
 10 A. Okay. I'll keep my mouth shut.
 11 (A luncheon recess was taken from 12:10 to
 12 1:10 p.m.)
 13 Q. You referred to an article by Angell and
 14 Kaiser, is that what it is?
 15 A. Kaiser. It was an editorial.
 16 Q. Who is Angell?
 17 A. It was Marcia Angell at the time. I think she
 18 was the editor of the New England Journal of Medicine.
 19 Q. Have you followed her work since she left the
 20 New England Journal of Medicine?
 21 A. Yes.
 22 Q. What has she been saying?
 23 A. She has had some comments about the industry.
 24 Q. Do you think she is a credible person?
 25 A. Yes.

1 web site, you can see that this is a treatment for
 2 cancer.
 3 Q. Um --
 4 A. They're saying treatment for cancer.
 5 Q. I want to clarify one thing. You said that you
 6 didn't hear it but you read it?
 7 A. I read the transcript.
 8 Q. You didn't hear the tape itself?
 9 A. I read the transcript.
 10 Q. I misunderstood that before. When you reviewed
 11 this material, how did you integrate this statement
 12 that appears on the web site that the information on
 13 this web site is not intended to diagnose a diagnosis,
 14 the information provided on the site is designed to
 15 support relationship that exists between patient's site
 16 visitor and his or her health care provider?
 17 MR. PAYNTER: I'm going to object. First ask
 18 him if he observed that when he was reading the web
 19 site.
 20 Q. Did you observe that? Do you recall observing
 21 that?
 22 A. I can't remember when I saw that, because I
 23 don't know when that appeared in their web site. Is it
 24 recent? I have no idea.
 25 Q. Let me ask you, did you see that statement when

1 Q. Was she critical of the drug industry?
 2 A. Yes.
 3 Q. Could you tell us some of the criticisms you
 4 remember?
 5 A. I can't remember them all but one was the
 6 pharmaceutical industry spends a great deal of time
 7 developing me too type drugs and not innovative enough.
 8 They spend too much money on marketing and advertising.
 9 Those are some of the things I remember.
 10 Q. Did she say anything about the quality of the
 11 studies done by the drug industry?
 12 A. I don't recall.
 13 Q. Do you think any of the things she said draw
 14 into question some of the outcomes of the studies that
 15 have been done by the pharmaceutical industry?
 16 A. I'm sure there were studies done by the
 17 pharmaceutical industry that were criticized and not
 18 perfect, yes.
 19 Q. You laid out the process that companies go
 20 through to get a product on the market.
 21 A. Yes.
 22 Q. Once they're on the market, does that mean
 23 they're home free and everything is fine?
 24 A. No.
 25 Q. Some of it may turn out not to be so good?

1 A. That's correct.
 2 Q. Has that happened in the cancer field?
 3 A. I'm not sure what you mean by not so good.
 4 Q. Did the FDA have to take drugs off the market
 5 that was previously approved?
 6 MR. PAYNTER: I'm going to object because he
 7 asked you to clarify.
 8 MR. J. TURNER: I asked what did the FDA say.
 9 MR. PAYNTER: That is another question than
 10 did the FDA remove something. He's asked you to
 11 clarify what you mean that some drugs were not so good.
 12 If you can please do that, but if you can't, please
 13 withdraw the question.
 14 Q. What I mean by not so good is that they pass
 15 tests and then turned out not to be able to remain on
 16 the market.
 17 A. You're specifically relating them to
 18 anticancer?
 19 Q. The first one I didn't but the second one I
 20 did.
 21 A. Can I talk about anticancer drugs?
 22 Q. Let's say without anticancer drugs.
 23 A. Have there been drugs withdrawn, yes.
 24 Q. Are there any anticancer drugs approved by the
 25 FDA that were subsequently withdrawn that you are aware

1 A. In the package insert of any drug, there's
 2 directions for its use. Or if you look at the PDR,
 3 physician's desk reference, for every drug listed there
 4 may be, not every one but for every drug there is a
 5 black box on top that is basically a warning.
 6 It then goes into this drug should not be given
 7 to patients who have had myocardial infarctions, heart
 8 attacks in the last six months because they may be at a
 9 greater risk. This drug should not be given to
 10 patients who have kidney dysfunction and there is a
 11 warning because after the drug was approved, additional
 12 patients who may have been excluded from the study were
 13 treated with the drug and low and behold they had some
 14 adverse effect.
 15 So there's warnings issued by the FDA to alert
 16 the farm -- physicians to be cautious with giving the
 17 drug or not giving it to certain patients at all.
 18 Q. The PDR pages, insert, is that a reprint of the
 19 package insert?
 20 A. Essentially.
 21 Q. Are there other warnings besides black box
 22 warnings within the PDR insert?
 23 A. Within the text of the use of the drug, in
 24 addition to describing what it's indicated for, what
 25 the doses are, how it should be given, formulated,

1 of?
 2 A. After they were approved, I'm not aware of any.
 3 Again, I'm specifically relating it to primary therapy
 4 of the cancer and not some supportive care agent.
 5 Q. Okay. Do you know of supportive care agents
 6 that have been approved by the FDA and then withdrawn?
 7 A. Not withdrawn but where the label was modified
 8 where warnings were put on it. That is the other thing
 9 that happens with drugs and is not surprising because
 10 there may be new adverse effects that occur in any new
 11 drug when the population of patients who are being
 12 treated is broadened beyond what was done in the
 13 clinical trial.
 14 So that should things -- some adverse effects
 15 of drugs may be uncovered until a much larger
 16 population of patients with many different other kinds
 17 of medication they're taking get exposed to it. What
 18 happens is when there are new side effects and
 19 everyone, very, very small percentage of patients,
 20 start developing those side effects, the FDA will issue
 21 what's called a black box warning and alert
 22 practitioners there may be additional concerns or tests
 23 they have to do or precautions they have to take in
 24 treating patients.
 25 Q. Okay. What is a black box warning?

1 there may be other warnings, other side effects and
 2 they usually list them all.
 3 Q. Okay. I want to now go to the part of the
 4 report "Detailed Discussion of Findings" and begin with
 5 Bio*Shark.
 6 A. Yes.
 7 Q. You began that by saying, "The key questions
 8 relating to Bio*Shark are: Does Bio*Shark inhibit
 9 tumor growth? Is Bio*Shark effective in the treatment
 10 of cancer?"
 11 A. Yes.
 12 Q. Who formulated those questions?
 13 A. Well, I formulated the questions in response to
 14 the scope of work on page four where I said I had been
 15 asked by the FTC to determine whether there is
 16 competent and reliable scientific evidence to support
 17 or substantiate the following claims, and the first
 18 one, does Bio*Shark inhibit tumor growth, and the
 19 second, Bio*Shark is effective in the treatment of
 20 cancer, and I turned it a -- I asked the question and
 21 addressed those questions with the available
 22 peer-reviewed literature that addressed whether or not
 23 Bio*Shark inhibits tumor growth and whether or not it's
 24 effective in the treatment of cancer.
 25 Q. You state that a number of reported

1 non-clinical studies suggested that a highly purified
 2 peptide isolated from shark cartilage may have
 3 antiangiogenic activity. Is that --
 4 A. That's correct.
 5 Q. Can you explain what that means?
 6 A. Well, do you want me to explain every word?
 7 Non-clinical study is not human, it's a test tube or
 8 animals. The highly purified peptides mean instead of
 9 taking crude shark cartilage, powdering it, chopping it
 10 up, they went through a biochemical process, very
 11 sophisticated biochemical process of actually purifying
 12 peptides or proteins that were within the shark
 13 cartilage. So they didn't just grind up the shark
 14 cartilage and throw it into the petri dish. They
 15 actually purified these proteins and then did tests in
 16 the test tube to see whether or not they could inhibit
 17 new blood vessel formation or angiogenesis.
 18 Q. Is there any shark cartilage that you're aware
 19 of on the market that you believe would meet standards
 20 that would allow it to perform in the way these studies
 21 described?
 22 MR. PAYNTER: Objection. No foundation. "Any
 23 shark cartilage"? There's no foundation. What is his
 24 experience with shark cartilage? There is no
 25 foundation for the question.

1 I can go into a health food store and get shark
 2 cartilage products in a health food store. If that's
 3 what you mean by "on the market."
 4 Q. Yes.
 5 A. But they're not highly purified.
 6 Q. You are saying -- I'm trying to understand --
 7 there are no, as far as you know, highly purified shark
 8 cartilage products on the market?
 9 A. That's right. Because they have been replaced
 10 by good antiangiogenic drugs that go after this
 11 process.
 12 Q. Can you tell me what some of those drugs are?
 13 A. Sure. There's a drug called Trastuzumab.
 14 Sorry about that. I always like the generic and its
 15 other name is Trastuzumab, T-R-A-S-T-U-Z-U-M-A-B, and
 16 its proprietary name is Avastin, A-V-A-S-T-I-N. Excuse
 17 me. It's name is Avastin, but its generic name is
 18 Bevacizumab. That's spelled B-E-V-A-C-I-Z-U-M-A-B.
 19 M-A-B at the end means monoclonial antibody, and that
 20 is Avastin. Bevacizumab is a synthetically generated
 21 monoclonial antibody. The target of Bevacizumab is a
 22 very important factor that stimulates new blood vessel
 23 growth.
 24 Q. And you said it stimulates?
 25 A. Stimulates, yes. The monoclonial antibody goes

1 Q. The question that I'm asking is regarding the
 2 statement "purified shark peptides" or whatever the
 3 word is that you used in that regard.
 4 MR. PAYNTER: Can we just let the record
 5 reflect accurately what he says. Just please read it
 6 accurately.
 7 MR. J. TURNER: Read it.
 8 A. "A number of reported non-clinical studies
 9 suggested that highly purified peptides isolated from
 10 shark cartilage may have antitumor activity and
 11 antiangiogenic activity," that is what I said. I
 12 didn't say crude shark cartilage. I said highly
 13 purified peptides from shark cartilage.
 14 Q. Are you aware of any shark cartilage products
 15 on the market?
 16 A. You have to tell me what you mean by "on the
 17 market."
 18 Q. Being sold to people who buy them.
 19 A. I'm not aware of highly purified peptides from
 20 shark cartilage on the market. I know about crude
 21 shark cartilage.
 22 Q. That is the question I asked you.
 23 A. I didn't understand it. I don't think you said
 24 purified peptides. You said am I aware of any shark
 25 cartilage on the market and that is different. I know

1 after the factor that stimulates new blood vessel
 2 formation and that factor is called V-E-G-F. It stands
 3 for vascular endothelial factor. So when the
 4 monoclonial antibody attaches, the VEGF stimulates it.
 5 So the stimulant for new blood formation is no longer
 6 there, so it inhibits new blood vessel growth. That
 7 drug, which is actually discovered by Genentech, is
 8 approved in the treatment of colorectal cancer with
 9 chemotherapy, approved in the treatment of non-small
 10 cell lung cancer and about to be approved in the
 11 treatment of breast cancer, always again with
 12 chemotherapy. Studies are on the way looking at it in
 13 brain tumors and other malignancies as well. That is
 14 just one.
 15 Q. Are you talking about just one or are you
 16 talking about two?
 17 A. Bevacizumab.
 18 Q. All one?
 19 A. So it's already approved with chemotherapy in
 20 the treatment of three different cancers and undergoing
 21 investigation for a number of others.
 22 There are other small molecules that go after
 23 the VEGF receptor, that is like a hormone, but the
 24 receptor is on a cell and when VEGF attaches to it, it
 25 sets into motion a series of biochemical reactions in

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1 the cancer cell, one of which is to turn on blood
 2 vessel formation or it inhibits the endothelial cells
 3 from multiplying and dividing and increasing new blood
 4 vessels.
 5 Q. Did you say it turns on?
 6 A. If you attach VEGF to the receptor, it sets
 7 into motion a series of biochemical reactions inside
 8 the cell. It could be in an endothelial cell. If you
 9 inhibit that by directing a chemical, small molecule,
 10 gets absorb, we know how much is absorbed, we know how
 11 much you need to inhibit new blood vessel formation, we
 12 know how much binds to the receptor, we know how long
 13 it stays on the receptors, we know it sets into motion
 14 these pathways and we also know it inhibits receptors
 15 and prevents all this from happening and there are a
 16 number of different drugs that do that.
 17 One is called Sunitinib, S-U-N-I-T-I-N-I-B.
 18 It's trade name is Sutent, and Sutent is made by
 19 Pfizer. And it's approved for the treatment of renal
 20 cell carcinoma and undergoing investigation in a number
 21 of other tumors. It is a breakthrough in the treatment
 22 of renal cell carcinoma.
 23 Another one is called Sorafenib,
 24 S-O-R-A-F-E-N-I-B, and its trade name is Nexavar,
 25 N-E-X-E-V-A-R, and Bayer makes that drug. It also is

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1 approved for renal cell carcinoma but also approved for
 2 the treatment of liver cancer for which there was very
 3 little before. So there are three different
 4 antiangiogenic medications but there are a number of
 5 others being evaluated today.
 6 Q. Are they redundant?
 7 A. No, not at all. If something is going after
 8 VEGF itself, that's completely different from Sunitinib
 9 or Sorafenib, which has different mechanisms of action,
 10 but one or more of the VEGF receptors is the target.
 11 Q. So a person that would be helped by the, let's
 12 just say, the Bayer drug might not be helped by -- did
 13 you say it was a Pfizer drug?
 14 A. Actually, people have been started on one or
 15 the other and switched over and have activity.
 16 Q. You said they were expanding the uses of those?
 17 A. Yes.
 18 Q. Do they have any side effects?
 19 A. Of course. Anything, every drug, whether
 20 pharmaceutical agent, or complimentary medicine,
 21 whether it's aspirin, it has side effects.
 22 Q. Do you know what kind of side effects these
 23 have?
 24 A. Yes.
 25 Q. What are they?

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1 A. Which one do you want me to start with?
 2 Q. Start with the same order that you did.
 3 A. The monoclonal antibody can cause high blood
 4 pressure. It may cause bleeding. It may cause
 5 allergic reaction because it's a monoclonal antibody.
 6 The Sunitinib may cause cardiovascular effects. The
 7 Sorafenib may also do some of that. It may have GI
 8 effects. But, again, some of these adverse effects can
 9 be graded in terms of their severity. If something
 10 causes nausea and vomiting, we have excellent agents
 11 that counteract the effect of a drug that causes that.
 12 Why should a patient suffer from an adverse effect that
 13 can be prevented or diminished so the drug is
 14 tolerable. Particularly if it improves survival of a
 15 patient.
 16 Renal cell carcinoma, if it spread to other
 17 parts of the body, up until recently it was very
 18 difficult to treat and Sunitinib now prolongs the
 19 survival of this disease.
 20 Q. Do you have any knowledge about how much it
 21 prolongs survival?
 22 A. Significantly prolongs survival by six months.
 23 Q. Six months?
 24 A. Yes.
 25 Q. Is that true of all three, about six months?

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1 A. Sunitinib has a better record in terms of
 2 overall survival. Bevacizumab has been very effective
 3 in prolonging time to tumor progression in colon
 4 cancer, lung cancer and breast.
 5 Q. When you say "very effective" --
 6 A. These are significant differences,
 7 statistically significant differences.
 8 Q. How much time would that add?
 9 A. It could be months.
 10 Q. How much did it cost to get each of these
 11 approved?
 12 A. I don't know.
 13 Q. Do you have an idea?
 14 A. I wouldn't guess.
 15 Q. Do you have an impression?
 16 A. I don't know what it cost Genentech, Bayer or
 17 Pfizer.
 18 Q. Do you think it's in the range of a hundred
 19 million dollars?
 20 A. I don't know the answer. I'm not going to
 21 guess what it costs them to do that, but it's
 22 expensive.
 23 Q. When you say "expensive," do you have a sense
 24 of what you mean by that?
 25 A. It may cost upwards of a hundred million

27 (Pages 102 to 105)

1 dollars from beginning to development and completion of
 2 approval for a new drug.
 3 Q. Do you have the table that analyzes the
 4 Bio*Shark studies?
 5 A. Yes.
 6 Q. Mine got --
 7 MR. PAYNTER: Let me give you that.
 8 MR. J. TURNER: I don't need it.
 9 Q. So I just wondered if you could give me a quick
 10 summary of that chart.
 11 A. Each of these studies listed here were clinical
 12 studies that were published in peer-reviewed journals,
 13 and actually were studies that had a study design that
 14 set out to show that some end point was going to be the
 15 primary end point of the study, and also in some of
 16 them established some secondary end points.
 17 For example, when you decide to do a study to
 18 show that drug X is better than placebo or that drug X
 19 plus chemotherapy is better than chemotherapy alone,
 20 you define, as I said, your patient population, what
 21 disease or diseases they have, what kind of prior
 22 therapy they have. They have to satisfy all of the
 23 eligibility criteria we talked about. You have to have
 24 a schedule of when you're going to administer the
 25 therapy. You have to have a base line evaluation to

1 concurrent therapy. So it wasn't -- they were getting
 2 shark cartilage alone versus concurrent therapy or
 3 shark cartilage alone plus concurrent therapy and
 4 concurrent alone. It wasn't a randomized study.
 5 So in that study it would be impossible to tell
 6 it could have been the treatment they were getting.
 7 Q. Was there a historical database on the
 8 treatment the patients got?
 9 A. You mean what kind of prior therapy did they
 10 have?
 11 Q. What I meant was there's the standard therapy
 12 plus shark cartilage being applied here.
 13 A. That's right, yes.
 14 Q. Is there any data on what the standard
 15 therapy's effects were in the historical database?
 16 A. I'm not following that.
 17 Q. So that product, whatever that standard
 18 treatment was, went through a phase I, II, III trial.
 19 A. Yes.
 20 Q. And did that establish a level of effectiveness
 21 of that product?
 22 A. Yes.
 23 Q. And the question I'm asking is: Was there any
 24 ability to compare the results that came when you added
 25 shark cartilage to it, to that historical record?

1 know where they're starting from in terms of having not
 2 only measurable disease but they have to have a disease
 3 that's been proven to be the disease you claim to be
 4 treating. Not that the patient says I have colon
 5 cancer, I would like to go on your study. We need to
 6 have the slides for the pathologist to review, medical
 7 imaging studies to know where the disease is to verify
 8 the fact that a patient has colon cancer and can go on
 9 the study. Patients are reliable but they don't have
 10 all of the information that's necessary to make a
 11 diagnosis and give them the best therapy that is
 12 available for them.
 13 Anyway, all of these were studies that have a
 14 predefined clinical end point; response, progression,
 15 free survival, time to tumor progression, progression,
 16 overall survival, quality of life. Those are the
 17 things we might look at. They're all listed here. A
 18 number of them are just case study, looked --
 19 Q. Study by Pruden?
 20 A. P-R-U-D-E-N. For example, case studies of
 21 patients who had different kinds of advanced metastatic
 22 cancer. He used a product called Catrux, which is
 23 actually Bovine, not shark cartilage, crude, not
 24 purified peptides, and he saw responses, complete
 25 responses in 19 patients but the patients had

1 A. You couldn't do it in that study because it
 2 wasn't controlled to look at what the standard of care
 3 was alone versus the standard of --
 4 Q. Let me ask it. Is there a way of finding out
 5 what the standard of care produces?
 6 A. Based on historical, yes, but it's not valid
 7 because you need to have what we call a concurrent
 8 control. You have to have patients being treated at
 9 the same time receiving the same kinds of medical
 10 imaging studies to avail the response, getting the same
 11 kind of supportive care. You can't take patients
 12 treated ten years ago and look at their results and
 13 throw in 31 patients treated ten years later and see
 14 how they did in comparison. That's not an acceptable
 15 clinical trial.
 16 You want me --
 17 Q. Just --
 18 A. Can I highlight some of them?
 19 Q. Highlight some of them.
 20 A. A little background. At Cancer Treatment
 21 Centers of America, as I mentioned to you earlier, most
 22 of the patients had a diagnosis of cancer. They had
 23 been treated before. Their disease invariably had come
 24 back and we found that many patients, I would say the
 25 majority of patients were taking some kind of

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1 alternative therapies or complimentary therapies that
 2 either somebody advised that they take or heard about
 3 it on the internet, they read about it in the health
 4 care magazine, their friends told them about it. We
 5 found that 70, 80 percent of patients are doing yoga
 6 and acupuncture and shark cartilage and coffee enemas,
 7 all these things they were self-administering and
 8 sometimes their doctors knew and often they didn't tell
 9 them because the doctors would get upset if patients
 10 were doing these things.
 11 We decided to do a study of shark cartilage,
 12 basically the same that William Lane had looked at in
 13 the patients in Cuba, and we decided to take patients
 14 who had been on prior therapy. They had a confirmed
 15 diagnosis of some advanced stage tumor, either lymphoma
 16 or other solid tumors, and the end treatment they were
 17 going to get would be shark cartilage, nothing else, no
 18 radiation therapy, only whatever general supportive
 19 care might be and our institution. It was very good
 20 supportive care, well nourished patients. They weren't
 21 randomized because what we were trying to do is, first
 22 of all, any evidence of activity, either tumor response
 23 or improvement in quality of life, after the first six
 24 weeks, if patients were tolerating the shark cartilage
 25 well, they would have their dose increase. It's dose

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1 escalation and we followed them with medical imaging
 2 studies every six weeks, and we were looking for
 3 primary end point which was evidence of complete or
 4 patient remission, improvement in quality of life and
 5 even stable disease.
 6 We plan to enroll a hundred patients in the
 7 study. We submitted the protocol to the FDA. They
 8 approved it, the cartilage product that we used was
 9 actually provided by a company who was selling it in
 10 the market. Actually, they gave us some support.
 11 Q. Was that a purified --
 12 A. No, none of these are purified. Not one of
 13 these things is purified peptide. They say partially
 14 purified. It's not purified and Bevacizumab doesn't
 15 have any -- whatever. It turns out after the first
 16 sixty patients were enrolled, we did analysis and we
 17 didn't see any evidence of response, no CRs, complete
 18 remissions, no partial remissions. There wasn't even
 19 improvement in quality of life. Inpatients who stopped
 20 their prior therapy, and we have an instrument to
 21 evaluate quality of life, I don't mean how do you feel,
 22 the patient says I feel great, that means nothing.
 23 There are instruments that patients can respond to,
 24 questions they respond to that can quantify whether
 25 their quality of life is better, the same or worse.

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1 All of this is objective. The important word
 2 that I'm trying to say today. Anyway, the bottom line
 3 is after the first 60 patients where we didn't see any
 4 responses, improvement in quality of life, we didn't
 5 see a decrease in prostate specific antigen level, in
 6 the men with prostate cancer, we elected to close the
 7 study.
 8 But it wasn't a controlled double blind
 9 randomized trial, but it didn't give us enough evidence
 10 to move evidence into a bigger study.
 11 I want to go down to Loprinzi at the Mayo
 12 Clinic, and they looked at Benefin, which was William
 13 Lane's shark cartilage product, and they did a phase
 14 III PC, which means placebo controlled, DB means double
 15 blind, and these were patients who got either Benefin
 16 or a placebo in what was considered the standard dose,
 17 although we really don't know, gram per kilogram per
 18 day of shark cartilage powder usually mixed with water
 19 or juice or something. They looked at inpatients with
 20 breast cancer and colorectal cancer. They looked for
 21 an improvement in response, and in the 42 patients
 22 studied, they didn't see any differences at all in a
 23 placebo controlled trial.
 24 Of all the studies listed here, I would range
 25 Loprinzi's as probably the best designed because it was

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1 a double blind placebo controlled trial.
 2 However, there was another study that I mention
 3 in my report, and it was a -- it was another randomized
 4 double blind placebo controlled trial with a product
 5 called Neovastat. It was made by a Montreal company
 6 called Aeterna, A-E-T-E-R-N-A. They claim that they
 7 patiently purified it, although it certainly wasn't the
 8 peptides that I talked about. They used a lower dose
 9 than the other shark cartilage studies and they did, as
 10 I mentioned, at MD Anderson Hospital in Houston, Texas,
 11 they looked at patients with non-small cell lung cancer
 12 that had tumors that could not be operated upon and
 13 treated with either standard chemo that we use today,
 14 which is taxane, T-A-X-A-N-E, that is standard therapy
 15 or chemotherapy and radiation therapy with either the
 16 Neovastats or placebo. They saw no differences. It
 17 did not improve overall survival and actually
 18 Neovastats has stopped the development, Aeterna stopped
 19 the development of Neovastats in cancer patients.
 20 That was presented at ASCO last year.
 21 Q. That is the one you said you reviewed after the
 22 chart?
 23 A. Yes. I got this summary from the NCI that was
 24 published in 2008 and left out of the new study so I
 25 added it here. So all of these studies are basically

1 the same, that none of them have shown any what I would
 2 consider reliable and competent data to suggest that
 3 shark cartilage, crude shark cartilage has any
 4 beneficial effect in a patient with cancer.
 5 Q. Okay.
 6 A. I can't say that about Bevacizumab, which is a
 7 monoclonal antibody. I can't say that about Sunitinib
 8 or Sorafenib or some of the epidermal growth factor
 9 epithelial growth factor, or some of the other drugs
 10 that actually go after a number of different receptors,
 11 because those all show real antiangiogenic activity,
 12 not only in the test tube, you can show it in patients.
 13 You can show a drug is decreasing blood flow by doing
 14 very interesting medical imaging studies and that's
 15 what you're looking for, evidence in the test tube that
 16 it's stopping new blood vessel formation causing
 17 shrinkage of tumors, causing stabilization of patient's
 18 clinical status and prolonging survival. That is what
 19 you're looking for.
 20 Q. All right. Let's go to 7 Herb Formula.
 21 A. Okay.
 22 Q. Again, the same question that I had for
 23 Bio*Shark is how were the questions that you're
 24 addressing formed?
 25 A. The same way I formulated the ones for

1 who?
 2 Q. What we have here is the label also indicates
 3 that each ounce contains two percent of the daily value
 4 of vitamin A and C. What I'm asking is you mentioned
 5 the label here. Do you know how the label for a
 6 product like this, the one we're discussing, is
 7 formulated?
 8 A. No. I have no idea. I just read the label. I
 9 don't know who designed it, who decided what to put on
 10 the label. This label doesn't actually tell me how
 11 much of the different seven major components are in it.
 12 It doesn't tell me how much burdock root, cat's claw or
 13 watercress is in the material. It says there is no
 14 calories, no carbohydrates, no proteins or fat. It's
 15 interesting because some of these products are
 16 carbohydrates and fats and have other ingredients.
 17 What the label says is in there and what the components
 18 are don't match either.
 19 Q. Say that again.
 20 A. The label says that 7 Herb Formula contains no
 21 calories, no carbohydrates, no protein, no fat, no
 22 cholesterol, no sodium. But let's take a look at
 23 Burdock root. It contains a number of different
 24 carbohydrates, fatty acids, volatile oils. Cat's claw
 25 contains glycosides and alkaloids and polyphenols.

1 Bio*Shark.
 2 Q. Okay. What kind of a product is 7 Herb
 3 Formula? Do you know what it is? Is it a --
 4 A. Well, I know that four of the ingredients in it
 5 were in another complimentary medicine that was
 6 developed in Canada by I think a nun. She spelled her
 7 name backwards to call it Essiac, and four of the seven
 8 ingredients in 7 Herbal or 7 Herb Formula were Essiac,
 9 Burdock root, cat's claw, sheep sorrel and Siberian
 10 ginseng. There are three additional products that DCO
 11 added to make 7 Herbal.
 12 Q. What are those?
 13 A. Slippery elm bark, Turkish rhubarb root and
 14 watercress.
 15 Q. Are you aware that it's tea?
 16 A. Now that you mention it, yes.
 17 Q. Okay.
 18 A. You drink it, is that what you mean?
 19 Q. Correct.
 20 A. Yes.
 21 Q. By the way, do you know how the labels for
 22 products like this are created?
 23 MR. PAYNTER: Objection.
 24 MR. J. TURNER: On what basis?
 25 MR. PAYNTER: Products like what? Labels for

1 There are a lot -- Siberian ginseng contains
 2 carbohydrates. It also polyenic acid. Those are fats.
 3 So what's in it doesn't match what the label says.
 4 Q. Are you familiar with the labels for tea?
 5 A. What kind of tea?
 6 Q. Any tea.
 7 A. I don't read the labels for tea. I don't drink
 8 tea.
 9 Q. Okay. You have comments on cat's claw. Tell
 10 us about cat's claw.
 11 A. It's alkaloids, comes from a tree. I'm not
 12 sure what the tree is called uncaria tomentosa,
 13 U-N-C-A-R-I-A, T-O-M-E-N-T-O-S-A.
 14 In vivo studies, again, with known doses of the
 15 material, I can't tell you what they were, I don't
 16 remember now, seem to have some effect on the immune
 17 response by increasing tea helper cell function and
 18 cells that gobbled other cells. Their function was
 19 increased and it seemed to inhibit some other factors
 20 that might have a negative effect on the immune
 21 response.
 22 It also had antiinflammatory activity, cut down
 23 on the inflammatory response which makes sense if it
 24 inhibits the tumor necrosis factor.
 25 It also had some side effects. Because when

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1 you gave it to patients who were taking medications for
 2 their blood pressure, it could cause low blood
 3 pressure. It could cause diarrhea. It also would
 4 cause bleeding and had an effect on the cells that
 5 helped the blood clot called platelets, so it would
 6 increase the risk of bleeding. So, again, there are
 7 immune effects but they're also side effects.
 8 Q. I want to go back to the labeling question.
 9 Are you familiar with the FDA regulations on labeling?
 10 A. I am familiar with the FDA requirements for the
 11 labeling of agents that I would use to treat cancer
 12 patients or new drugs that are approved.
 13 Q. Are you saying as a professional opinion that
 14 the label for 7 Herb Formula violates labeling
 15 regulations?
 16 A. I don't know the answer to that. That's not
 17 for me to decide. Some of the other products do have
 18 the amount of material in them. They give you the
 19 number of grams or milligrams of different components
 20 for a lot of these, but what was interesting with 7
 21 Herb Formula, it's got the seven components but there's
 22 no how much of it is in there and I couldn't find out
 23 anywhere how much is in there because I wanted to know
 24 if I were to correlate the non-clinical studies where
 25 specific amounts of some of these materials were added

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1 to test the activity, I didn't know how much was in
 2 herb formula of the comparable materials to know how
 3 close it came to the experimental conditions.
 4 There is a dose response effect in medicine, in
 5 pharmacology. As a certain dose you don't see any
 6 effect. At another dose you might see the effect
 7 you're looking for. Sometimes you increase the dose
 8 and might see a reversal of that effect. There's
 9 always a dose response for not only activity and
 10 efficacy, but there's dose response for toxicity. It
 11 would be important to know if you're comparing these --
 12 ingredients in 7 Herb Formula to compare it to what is
 13 in the published literature about the activity of these
 14 different components.
 15 Q. I noted that on Siberian ginseng you cited
 16 Cassileth and Lucarelli.
 17 A. Again, Cassileth and Lucarelli is not a peer
 18 reviewed article. It goes over all of the different
 19 herbals that are available, not 100 percent but there
 20 are many in there. They describe what's in it, how it
 21 works, if a mechanism of action is known, whether there
 22 are any interactions with other anticancer drugs, what
 23 the non-clinical data are and, if available, any
 24 clinical studies to support their use in treating
 25 cancer patients.

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1 Q. Do you know if they wrote about Burdock root?
 2 You didn't cite it for Burdock root.
 3 A. Burdock root is in their book. It's in their
 4 book.
 5 Q. How about cat's claw?
 6 A. That is in the book.
 7 Q. Was there a reason why you cited them on
 8 Siberian ginseng but not on the others?
 9 A. No intent. I know I reviewed Cassileth and
 10 Lucarelli for all of these ingredients in 7 Herb
 11 Formula. I can't tell you why I cited them for --
 12 perhaps maybe I couldn't find a primary reference to
 13 support the stimulation of tea lymphocytes and natural
 14 killer cells has been reported, but the mechanism of
 15 immunostimulation is unknown. And I think it was the
 16 last part, the mechanism of the immunostimulation is
 17 unknown, came from something that Cassileth and
 18 Lucarelli said in their section on Siberian ginseng.
 19 Q. Those are the four in the basic product, right?
 20 A. Yes.
 21 Q. And then the other three, let's see, slippery
 22 elm?
 23 A. Yes.
 24 Q. What were the other two, Turkish rhubarb?
 25 A. Turkish rhubarb root and watercress.

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1 Q. Are those in the Cassileth book?
 2 A. Yes.
 3 Q. And so then the only one of those seven that
 4 you cited was the Siberian ginseng?
 5 A. That's correct. But, for example, under
 6 watercress references are cited and those studies I
 7 know were in the Cassileth and Lucarelli section on
 8 watercress.
 9 Q. Is Turkish rhubarb a food or a drug?
 10 A. What are you using it for? Are you using it to
 11 treat cancer, then it's a drug. If you're using it as
 12 a supplemental to your diet or complimentary medicine
 13 to cancer therapy and not making any claims that it has
 14 anticancer activity and increase response to
 15 chemotherapy or prolong your survival, if that's all
 16 you're saying it would be, in my mind it's
 17 supplemental.
 18 Q. Supplement.
 19 A. Yes.
 20 Q. I noted when I read through here I didn't find
 21 any place where you mention supplement. I may have
 22 missed it. You never talked about any of these things
 23 as a supplement.
 24 A. Somewhere in this report I say if these things
 25 are being used to add to but not replace proven

1 efficacious therapy that's based on reliable and
2 competent data, then to me that's complimentary
3 medicine, a supplement to what you're taking, but it's
4 not a replacement for.

5 Q. Are you familiar with the concept dietary
6 supplement?

7 A. Sure.

8 Q. How are you familiar with that?

9 A. They're recommended daily amounts, daily
10 requirements for a number of different vitamins,
11 minerals, iron, vitamin B, D complex and without those
12 over a period of time, one can become deficient and
13 suffer some of the metabolic effects of deficiency.

14 Q. How about herbs, are they a dietary supplement?

15 A. Depends how they're being used. I have no
16 argument with someone saying we would like to add these
17 things to conventional chemotherapy because we think it
18 might make you feel better. We don't want it to
19 replace, we're not making a claim it can cure your
20 cancer or stop your tumor growth, but we think it might
21 be helpful and not harmful. I have no argument with
22 that, but don't tell me that this can take the place of
23 treating your breast cancer because whatever.

24 Q. Do you believe that these products, each of the
25 ones that you're looking at, four of them, are

1 Irinotecan. I don't know the answer to that. The
2 point is there is a warning now don't take St. John's
3 Wort with this because it will decrease the beneficial
4 effect of the therapy.

5 Q. Where is that warning, is it with St. John's
6 Wort or --

7 A. With the chemotherapeutic agent.

8 Q. Are you familiar with warnings on other drugs
9 like that?

10 A. Yes.

11 Q. Tetracycline?

12 A. Yes.

13 Q. What is the warning?

14 A. I don't know what the warning is.

15 Q. Don't take it with cheese and dairy products?

16 A. I don't know. Virtually every drug in its
17 package insert or label, like PDR, will have warnings
18 about what it may interact with. There are drug
19 interactions with most drugs now that when we're
20 developing a new drug we are very concerned about
21 certain kinds of other medications that many people
22 take that can interfere with the metabolism of the drug
23 we're testing.

24 Two things can happen. The drug you might be
25 taking for a seizure disorder or a drug you might be

1 dangerous?

2 A. They could be for some of the reasons why I
3 talked about where some of them may cause side effects.
4 Some of these agents might interfere with effectiveness
5 and decrease their activity. So they could potentially
6 be dangerous. We know that's true.

7 I'm sure you heard of complimentary medicine
8 called St. John's Wort, W-O-R-T. And we now know that
9 St. John's Wort contains chemicals that actually
10 counteract the anticancer effects of a very effective
11 chemotherapeutic agent.

12 Q. What is that?

13 A. Camptothecin, C-A-M-P-T-O-T-H-E-C-I-N. That is
14 the class. The drug would be Irinotecan,
15 I-R-I-N-O-T-E-C-A-N. It is used in colon cancer, can
16 be used in lung cancer, might be used in breast cancer.

17 Q. Who manufactures that drug?

18 A. The Camptothecins?

19 Q. Yes.

20 A. Couple of them out there. I think Pfizer makes
21 one. I'm not sure about the other.

22 Q. Those are FDA approved?

23 A. FDA approved.

24 Q. So that costs maybe \$100 million?

25 A. I don't know how much it cost to develop

1 taking for hypertension may block the breakdown of the
2 chemotherapeutic agent. It blocks its metabolism. So
3 you convert it from something active, potentially toxic
4 for something that hangs around for a longer period of
5 time and you get toxic effects.

6 There may be other drugs that speed up the
7 process of metabolizing a drug. What happens is if you
8 break it down faster, you never get a level of the drug
9 in your body that's going to be beneficial.

10 A lot of people take blood thinners, we call
11 them Warfarin, W-A-R-F-A-R-I-N, to prevent clots and
12 sometimes the interaction of a drug and the drug you're
13 taking is such that you may get higher levels of
14 Warfarin that cause you to bleed. So we have to always
15 know what these what we call drug interactions are and
16 it's a very important part of the process of drug
17 development.

18 Q. What I was asking you is there are similar
19 things about drug food interactions?

20 A. Some drugs may not be absorbed on a full
21 stomach. Others it doesn't make any difference. Part
22 of the evaluation of every new drug is to do the study,
23 giving it to healthy volunteers, sometimes if it's not
24 a cancer drug or to cancer patients either on an empty
25 stomach or food to see if there is any difference in

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1 absorption.
 2 We do studies in patients with known kidney
 3 trouble to see if there is a difference in the
 4 metabolism. Safety evaluation is designed to protect
 5 patients. We can't study the drug interactions for
 6 every drug out there that has treated a lot of other
 7 disorders, like diabetes, hypertension, some of the
 8 statins used to treat high cholesterol levels, many
 9 American men for erectile dysfunction, but there may be
 10 interactions where someone is taking an erectile
 11 dysfunction drug and is on a chemotherapeutic agent and
 12 may not be tested in the earlier phases but it's
 13 possible one of the newer drugs might interact with one
 14 of these drugs.
 15 We know there are problems with patients who
 16 are on medications for high blood pressure that you
 17 hear every time on television and listen to one of the
 18 advertisements but not all the side effect are
 19 described. It should be in the label or package insert
 20 but sometimes we discover new side effects that we
 21 never encountered before.
 22 Q. How do those get into the labels?
 23 A. People are obliged to report adverse events
 24 even after a drug has been approved and marketed.
 25 These are post approval safety reporting. The

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1 companies are responsible for reporting it and if there
 2 is a trend, there are signals now if the new drug and
 3 some other agent is causing serious problems. There
 4 are warnings put out and eventually it gets into a
 5 black box.
 6 Q. How effective is that adverse reporting system?
 7 A. Doctors get letters and new results show there
 8 is a bad interaction with our new drug and patients on
 9 some other kind of drug, and be careful when you give
 10 it. Watch this. Do these tests.
 11 Once its been reported and someone pays no
 12 attention to it and this patient has some horrible
 13 adverse effect because she decided not to follow the
 14 advice, a patient would certainly have a recourse to
 15 sue the doctor for malpractice.
 16 Q. Talk a little bit about your report on Turkish
 17 rhubarb root.
 18 A. Okay.
 19 Q. Just describe it.
 20 A. Here's an interesting situation where different
 21 doses cause different effects. At low doses, again,
 22 these are specific doses now, we don't know what the
 23 dose is in 7 Herb Formula but at low doses, the rhubarb
 24 root tannins cause constipation and at higher doses,
 25 two other metabolites ingredients can cause diarrhea.

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1 One dose level you have constipation and a higher dose
 2 level is diarrhea. That is important to know how much
 3 is in there, what are the effects of doses being given,
 4 how much is being absorbed and what other interactions
 5 there may be.
 6 There have been some studies in mice to show
 7 antitumor effects but, again, I say this over and over
 8 again. No studies have been performed in humans with
 9 cancer, thus there is no supporting data. Because it
 10 worked in a mouse, doesn't mean that it's going to work
 11 in a human. We can cure cancer in mice. We can put
 12 pancreatic cancer cells into the behind limb of a
 13 little white mouse and treat it with different chemo
 14 agents and make the tumor disappear. Because I cure
 15 that mouse of pancreatic cancer that's from a human,
 16 can I cure pancreatic cancer in people? Five percent
 17 are surviving for a few years. We don't have any
 18 effective therapy. So even though it works in a mouse,
 19 I can't make that huge leap across the Grand Canyon of
 20 clinical research and say because it worked in a mouse,
 21 a nude mouse that has no immune or carefully
 22 genetically engineered mouse, I can't say because it
 23 worked in a mouse it will be efficacious in man. Can't
 24 say it. Otherwise you wouldn't have to do phase I, II,
 25 III studies. We do study in the mouse, see some tumor

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1 response and we can approve the drug. Won't work.
 2 Very dangerous.
 3 Q. With regard to watercress, describe your
 4 discoveries on watercress.
 5 A. Watercress seems to be an agent or components
 6 seem to be an agent that may have some benefit in
 7 urinary tract infections in children or bronchitis or
 8 even parasites that are invading the liver. Those were
 9 the studies of Hecht. It's not clear whether it's an
 10 irritant of mucous membranes or might reduce
 11 inflammation, so it's confusing, but there was a study
 12 again by Hecht, who seems to be the individual looking
 13 at watercress more than anybody else, in an animal
 14 model. He believed he could show the decrease in the
 15 production of a carcinogen that is present in tobacco
 16 smoke.
 17 Bottom line, there are no clinical studies to
 18 show any of these effects in either cancer treatment or
 19 cancer prevention. Patients were to chew watercress
 20 leaves and they were smokers, it would be interesting
 21 to show that in man you can decrease the formation of
 22 certain carcinogens that are present in tobacco smoke
 23 and smokers. If that were the case, you might be able
 24 to prevent lung cancer in smokers. Better thing would
 25 be to have them stop smoking but, again, there is just

1 not enough information to say that watercress will
 2 prevent cancer in a human being.
 3 Q. Is watercress a food or drug?
 4 A. I thought watercress was something I put in my
 5 salad. It's food.
 6 Q. Food?
 7 A. Again, you don't chop up watercress and put it
 8 in the test tube or give it to animals. You take the
 9 active ingredients. That's really what we should do.
 10 It's not the leaf. It's what's in the leaf in a
 11 certain amount that may be active.
 12 If you look at my table there are glycosides in
 13 watercress that may be the active ingredients that are
 14 having these effects on the generation of cancer
 15 causing chemicals.
 16 Q. I have the same question about the original
 17 four items that were in the first formula. Burdock
 18 root, is that in your opinion a drug or a food?
 19 A. Depends on how you're using it for the reason I
 20 gave.
 21 Q. Then Siberian ginseng?
 22 A. Again, Burdock root, let's look at Burdock
 23 root. What's in there? What does Burdock root have
 24 that might have some activity, flavonols and
 25 polyphenols, which is quercetins, and I think have some

1 Q. Can you describe what a pharmacologic effect
 2 is?
 3 A. Everything we take, any medication we take has
 4 an effect on some organ or tissue or metabolic pathway
 5 in our body and these are usually measurable. Simple
 6 example is aspirin, very widely used, but why do people
 7 who have had a heart attack take a baby aspirin every
 8 day or if they had a stroke. Low dose of aspirin
 9 readily absorbed by the body has the ingredient, active
 10 ingredient of acetylsalicylic,
 11 A-C-E-T-Y-L-S-A-L-I-C-Y-L-I-C, acid which binds to
 12 platelets. And platelets are sticky little cells that
 13 can clog up blood vessels. You've seen the
 14 advertisements for Plavix on television. If you can
 15 inhibit, block the ability of platelets from sticking
 16 together, you can prevent clot formation in blood
 17 vessels like arteries and you can protect people from
 18 developing another stroke or heart attack.
 19 So the pharmacological activity is that a
 20 certain dose of aspirin will have a specific effect on
 21 the function of platelets and you can measure that.
 22 You can see how sticky they are. You can test
 23 different doses of whatever drug it might be against a
 24 laboratory test of platelet function and you can see
 25 the pharmacological effect. It's dose response effect.

1 nutritional value. There have been studies to suggest
 2 that some of them may have anticancer activity in the
 3 laboratory. So I'm not opposed to those things but,
 4 again, how much is in there and how much of the Burdock
 5 root flavonols get absorbed and get absorbed in an
 6 amount that might have a beneficial effect. If you
 7 look at what is inside the Burdock root, you have to
 8 look at the active ingredient that will have an effect
 9 on cancer cells, cancer prevention.
 10 Q. Some --
 11 A. But if you're only using it to make people feel
 12 better and not stating this is to be used to treat your
 13 cancer or you can use it with your conventional cancer
 14 therapy and it's going to make it better, make the
 15 therapy better, I have no problem with that, if you
 16 have evidence to prove it. I want competent and
 17 reliable data to show if I gave a patient with
 18 non-small cell lung cancer the active measurable
 19 amounts of ingredients in Burdock root along with
 20 chemotherapy and they tolerated chemotherapy better,
 21 they had a better response rate, progression of time to
 22 tumor progression and I had a randomized trial to show
 23 the Burdock root plus the chemo is better than chemo
 24 alone, I wouldn't have any problem at all saying I
 25 don't have a problem with this.

1 Q. Do foods have pharmacologic effects?
 2 A. Depends on what food it might be.
 3 Q. Can you give an example of a food that has
 4 pharmacologic effects?
 5 A. Orange contains vitamin C.
 6 Q. So you would say that vitamin C does have
 7 pharmacological effects?
 8 A. Of course.
 9 Q. Do all vitamins?
 10 A. Yes.
 11 Q. And all minerals, do they have --
 12 A. All minerals?
 13 Q. Yes. Let's just talk about minerals that we
 14 consume as food.
 15 A. Lead is a mineral. I'm not sure it has a very
 16 good effect. I wouldn't recommend it.
 17 Q. Are all pharmacologic effects positive?
 18 A. No.
 19 Q. Lead effect, is that a pharmacological effect?
 20 A. Sure. It causes brain damage and all kinds of
 21 terrible things but most vitamins that we have minimum
 22 recommended amounts have a beneficial effect because --
 23 Q. But that is a pharmacological effect, is that
 24 what you're saying?
 25 A. Yes. What would we take it for? Why would we

1 take something if it isn't going to have a
 2 pharmacological physiological beneficial effect.
 3 Q. So are you saying that all effects of foods are
 4 pharmacologic effects?
 5 A. No. Some are purely nutritional and giving you
 6 calories.
 7 Q. That is what I was trying to make a distinction
 8 on. Caloric effects are not pharmacological?
 9 A. In having a specific mechanism of action, no.
 10 Q. So --
 11 A. We need calories in our diet. We need sugar,
 12 proteins, which are building blocks to help our body
 13 make protein, and there are other things that have
 14 specific biochemical or pharmacological effects on
 15 other pathways.
 16 Take iron. If we didn't have any iron in our
 17 diet and let's say we had early stage colon cancer and
 18 losing blood every day, we didn't know it over a period
 19 of time we would become iron deficient and anemic.
 20 Iron is present in some foods. All we can take is a
 21 supplement of iron, tablet. So those things are
 22 vitally important.
 23 If we don't have vitamin B12 in our diet, we
 24 can develop neurological problems or severe anemia,
 25 though cease to have important roles to play in normal

1 A. Yes, I do.
 2 Q. Is this the label you looked at?
 3 A. Mine was in black and white but it was the
 4 label I looked at.
 5 Q. You indicate that bromelain and boron --
 6 because the amounts of bromelain and boron are not
 7 provided in the label, daily amount of these
 8 ingredients is unknown. Can you find that?
 9 MR. PAYNTER: We haven't actually reached GDU,
 10 have we? I think you were just finished up --
 11 MR. J. TURNER: We were finishing up 7 Herb
 12 Formula.
 13 MR. PAYNTER: I don't think you started it.
 14 A. I see that. The only thing I can say since I
 15 put down the quantities of every other material, I just
 16 can't recall whether -- I didn't have a colored label.
 17 I had a black and white one. I'm not sure whether it
 18 was the same one, and when I say I don't know the
 19 amount of bromelain and CDU, according to this label
 20 there are -- I can't read it. My glasses are not good
 21 enough. Is it 20,000?
 22 Q. I think it's 2,000?
 23 A. 200,000?
 24 Q. 2-O-O-O.
 25 A. According to this label the amount of bromelain

1 physiology.
 2 (A recess was taken.)
 3 A. You had asked me in the discussion of 7 Hearing
 4 Formula why I had only cited Cassileth and Lucarelli
 5 under one of the ingredients, but actually in my table
 6 two, I have the constituents of 7 Herb Formula which
 7 lists the constituent and carbohydrate content, fat,
 8 cholesterol and other ingredients. And all of that
 9 came from the Cassileth and Lucarelli sections on each
 10 of the different compounds because that's how she
 11 organized her sections. So I did rely on it for other
 12 ingredients besides the one we talked about.
 13 MR. J. TURNER: Okay. I'd like this to be
 14 marked as our first exhibit, wherever we are in -- we
 15 don't have any.
 16 MR. PAYNTER: We don't have any, so this is
 17 number one.
 18 MR. J. TURNER: I think maybe one and only.
 19 (Labels for each of the four products were
 20 marked as DCO Exhibit 1 for identification; 2-6-09,
 21 L.S.)
 22 Q. I've given you DCO 1 which is the labels of
 23 each of the four products. I'm actually directing your
 24 attention to the GDU label. Do you recognize that
 25 label?

1 in a serving is listed on this label. I just don't
 2 remember whether the one I had had it, because I know I
 3 would have included it because it was very important
 4 for my discussion.
 5 Q. Is there a way that we can ascertain whether
 6 his label he reviewed had the numbers on it or not?
 7 MR. PAYNTER: I certainly can go back and look
 8 and see what we sent him.
 9 MR. J. TURNER: Can we?
 10 MR. PAYNTER: Now?
 11 MR. J. TURNER: No, at some point.
 12 MR. PAYNTER: I believe whatever we sent him
 13 were labels we received from the company in the course
 14 of the investigation. Maybe at some point it wasn't on
 15 there, but in any rate we can check.
 16 A. The fact that I didn't --
 17 MR. PAYNTER: There is no question.
 18 Q. Let me go back to the first GDU question, which
 19 is how were the questions that you addressed
 20 formulated?
 21 A. Exactly the way the other sets were formulated.
 22 Q. Could you describe the ingredients of GDU as
 23 you understand them?
 24 A. Yes. The components of GDU are bromelain,
 25 which is a proteolytic enzyme. And it also has an

1 enzyme that breaks down clots, called fibrinolytic
2 enzyme. The next ingredient is curcumin, that's
3 polyphenol. The next ingredient is Quercetin,
4 Q-U-E-R-C-E-T-I-N, which is a plant flavanoid. The
5 next one is Fever Few. The important thing about Fever
6 Few is its active ingredient is Parthenolide,
7 P-A-R-T-H-E-N-O-L-I-D-E. Those are the -- then it has
8 boron.

9 It also contains what is called a biomolecular
10 base, which is listed on the label and contains a
11 number of different ingredients. I can't read this
12 without a magnifying glass but I read it before. I
13 used my magnifying glass to read it.

14 Bromelain, tumeric, quercetin, Fever Few, boron
15 and then the biomolecular base which contains a lot of
16 vitamins, minerals, elements.

17 Q. Have you discussed that base earlier in the
18 report? I'm not sure if this is the place where it
19 says "as discussed earlier," but I'm just --

20 A. I think I discuss it -- is it in 7 Herbs or
21 BioMixx.

22 Q. BioMixx is next.

23 A. I'm just trying to think of where else it was.

24 MR. PAYNTER: I think it must be 7 Herb.

25 A. No. I think it should be below. I may have

1 A. Under the section on tumeric curcumin.
2 Q. There's the beginning of a sentence which says
3 tumeric, curcumin in parenthesis. The question is:
4 Are you saying tumeric and curcumin are the same thing?
5 It's after bromelain.

6 MR. PAYNTER: Can you repeat your question?

7 Q. Yes. That it's after the section on bromelain
8 there is another section tumeric (curcumin) and I'm
9 asking are you saying tumeric and curcumin are the same
10 thing?

11 A. I'm not sure if they're exactly the same thing
12 but I was using them interchangeably because I think
13 the active material here is curcumin, which I think is
14 in tumeric. I'm just not sure if they're exactly
15 interchanged.

16 MR. PAYNTER: Can you let him answer the
17 question?

18 A. I'm not sure if they're interchanged, when you
19 talk about tumeric you're really talking about
20 curcumin, and most of the studies that I refer to have
21 been studies of curcumin rather than tumeric. If you
22 look at the titles of the papers and what was
23 evaluated, it was curcumin in those papers.

24 Q. Do you know how many single agents there are in
25 tumeric?

1 changed the order of this. It should be as discussed
2 below in BioMixx.

3 Q. We'll talk about it there. You indicate that
4 tumeric or curcumin is the single most promising agent
5 in the products you looked at.

6 A. Correct.

7 Q. What do you mean by "promising agent"?

8 A. Well, again, based upon peer-reviewed
9 literature, both non-clinical and clinical studies,
10 curcumin appears to be an agent warranting further
11 study for two reasons. It may actually be a cancer
12 preventive agent, particularly in colorectal cancer,
13 for example, patients who may have polyps and it may
14 have an antitumor effect.

15 Again, these are preliminary studies, but I
16 think the available data today would suggest that it
17 would warrant further investigation. Again, it's based
18 on peer-reviewed literature, clinical trials and
19 non-clinical studies.

20 Q. When you introduced that concept, you say
21 tumeric and then in parentheses curcumin, do you see
22 where that is in your report?

23 A. No.

24 Q. It's right -- we start GDU -- I have it but he
25 doesn't. We have to try and get him to that point.

1 A. How many different agents there are? I don't
2 know exactly.

3 Q. You indicate that it has a long history of
4 traditional Indian and Chinese medicine to treat
5 inflammatory diseases, abdominal disorders and other
6 ailments, including cancer?

7 A. Yes.

8 Q. How did you learn that set of facts?

9 A. From papers on curcumin as well as treatises,
10 like Cassileth and Lucarelli. Very often in a paper on
11 curcumin, background, historical background might be
12 included in the introduction of a paper. And some of
13 the papers on studies in curcumin, for example, the --
14 let me give you a specific citation.

15 The reference section on GDU references there's
16 a paper by Huang, et al, 1994, "Inhibitor effects of
17 dietary curcumin on forestomach, duodenal, colon
18 carcinogenesis in mice."

19 Paper by Jiao, "Curcumin, a cancer
20 chemopreventive and chemotherapeutic agent, is a
21 biologically active iron chelator. Blood 2009," just
22 published. Very interesting paper because curcumin
23 actually binds with iron and may cause iron deficiency.
24 Just published a few weeks ago.

25 Another paper by Kawamori, "Chemopreventive

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1 effect of curcumin."
 2 I don't know one by Rao, "Chemoprevention of
 3 colon carcinogenesis by dietary curcumin." So all of
 4 these papers that I've cited, talk about dietary
 5 curcumin. Some of them they may have mentioned where
 6 they came from, what the historical background was, but
 7 that is where that statement came from. All of these
 8 published papers and peer-reviewed literature use the
 9 term curcumin, not tumeric.
 10 Q. You make the statement concerning lacking
 11 double blind placebo controlled randomized clinical
 12 trials of curcumin. Could you summarize your -- the
 13 significance of that section in which you talk about
 14 the lack of those studies?
 15 A. Before I got to that sentence I described what
 16 are the reported studies and what some of the results
 17 were of those studies, particularly some of the studies
 18 in patients who are at high risk of developing colon
 19 cancer, but the ultimate step to demonstrate in a
 20 competent and reliable way that curcumin actually does
 21 these things would be to do a double blind placebo
 22 controlled randomized clinical trial. That's how we do
 23 things to show that it really is effective.
 24 Q. Effective?
 25 A. In preventing cancer or treating colon cancer.

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1 Q. Right under that then is the section on
 2 Quercetin?
 3 A. Yes.
 4 Q. Describe that section and what its significance
 5 is.
 6 A. This is a flavanoid. It is a number of things
 7 we eat or drink, like apples, tea, onions, buckwheat.
 8 The non-clinical studies are to show it has a number of
 9 different actions, cutting down on inflammation or
 10 being antioxidant or actually cutting down on allergic
 11 reactions. There have been some proposed mechanisms of
 12 action in a number of different areas that are
 13 important in cancer cells, like this P53 gene is
 14 important because if it's abnormal it doesn't shut down
 15 cancer cells.
 16 In other non-clinical studies it may cause
 17 cells to stop multiplying and dividing. It can inhibit
 18 certain important metabolic enzymes, tyrosine,
 19 T-Y-R-O-S-I-N-E, kinase. It can also block the binding
 20 of estrogens to the receptor which might be important
 21 in breast cancer.
 22 Heat-shock proteins are additional agents that
 23 can cause tumor cells to die. And if it blocks the
 24 expression of certain genes that are important in the
 25 cancer process, that might be beneficial also.

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1 But in summary, although these are proposed
 2 mechanisms of action mostly from non-clinical studies,
 3 we are again lacking any randomized clinical trials in
 4 quercetins alone, purified set dose in cancer patients
 5 to show that it has beneficial effects.
 6 Q. When you say to show it has beneficial effects,
 7 what do you mean by "beneficial effects"?
 8 A. I discussed some of those end points that can
 9 be evaluated. Does it, when given with anticancer
 10 therapy, improve response rates? Does it prolong the
 11 time to tumor progression? Does it prolong survival?
 12 Does it improve the quality of life? Does it increase
 13 the tolerance to conventional chemotherapy without any
 14 added toxicity? Those are all reasonable end points
 15 that one would look at to see whether or not something
 16 is effective as an anticancer treatment.
 17 Q. Then the next thing is Fever Few?
 18 A. Yes.
 19 Q. Could you describe Fever Few the way we did --
 20 A. As I state in my report, the major active
 21 ingredient in Fever Few is a chemical called
 22 parthenolide, P-A-R-T-H-E-N-O-L-I-D-E. A number of
 23 non-clinical studies have been done and they show, for
 24 example, in colon cancer it induces a programmed cell
 25 death, very important process in causing cancer cells

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1 to die.
 2 There's been an open label non-randomized phase
 3 I study of Fever Few, actually a proprietary form of it
 4 called Tanacet, T-A-N-A-C-E-T. And this was a
 5 condition in cancer patients and they started off --
 6 you usually do in a phase I study, as I mentioned
 7 earlier today, you do dose escalation, start off with a
 8 low dose and after a few patients are treated with a
 9 low dose and you don't see any dose limiting toxicity,
 10 you escalate the dose to another level and then another
 11 level and another level.
 12 In this study they treated 12 patients. The
 13 males had prostate cancer and the single female had
 14 breast cancer. They had measurable disease. They had
 15 defined performance status. They had a life expectancy
 16 of greater than three months. They were going to
 17 evaluate response by predefined criteria at set
 18 intervals and they were hoping to identify a safe and
 19 active dose, and they also did pharmacokinetic studies
 20 and they only administered Fever Few in these patients.
 21 I must say it's not necessary to show efficacy
 22 in a phase I study. You need to show what is the
 23 maximum tolerated dose and the safety profile and
 24 what's the dose we can use in phase II where you want
 25 to evaluate response or other end points of efficacy.

1 That wasn't done.
 2 They did find that in the patients who were
 3 given the parthenolide, they couldn't measure any of
 4 the compound in the circulation. It was given by
 5 mouth. And either it wasn't absorbed very well or what
 6 was absorbed was so low that it was below the level of
 7 detection by biochemical tests they used to measure it.
 8 It's not possible to say anything from this study
 9 because they never did get to the maximum tolerated
 10 dose, so that before you can say whether Fever Few is
 11 active in cancer patients, you have to do more studies
 12 with purified parthenolide, which is the admitted
 13 addictive ingredient here.
 14 We don't know anything at all about Fever Few
 15 yet. We don't have complete pharmacokinetic studies.
 16 We don't have pharmacodynamic studies. MTD was never
 17 established so we don't know what its full safety
 18 profile is.
 19 But it's interesting, you've asked me this many
 20 times today, are there side effects of these things,
 21 yes. Even at these extremely low doses where the
 22 amounts of parthenolide in the patients was so low it
 23 couldn't be detected and they were only getting
 24 parthenolide, there were a number of different side
 25 effects seen; fever, nausea, diarrhea, indigestion,

1 a capsule or serving, what is it?
 2 What I saw, and it's in my report and I took it
 3 from the label, I didn't make it up, I took it from the
 4 label that talked about recommended numbers of capsules
 5 a day. And the recommended DCO recommended daily dose
 6 of GDU, and this came from the label I saw said three
 7 to six capsules, two to four times per day. That would
 8 be a total of six to 24 capsules a day.
 9 Based on the label I saw, the amount of Fever
 10 Few would be then 600 to 2,400 milligrams because each
 11 serving or capsule, I can't tell, it's not clear, is a
 12 serving capsule or three capsules, that total would be
 13 600 to 2,400 milligrams of Fever Few a day.
 14 MR. PAYNTER: I just want to ask you where did
 15 you get this label?
 16 MR. J. TURNER: We got them from Daniel
 17 Chapter One.
 18 MR. PAYNTER: Because we did -- we produced to
 19 you what you guys produced to us, so those would have
 20 been more appropriate to us because we never received
 21 these.
 22 MR. C. TURNER: You can get the label.
 23 MR. J. TURNER: You say we have it because
 24 you've given it to us.
 25 MR. PAYNTER: Yes, in our production to you we

1 fatigue and blurred vision at the lowest dose.
 2 Q. Was this study done on Tanacet?
 3 A. Yes.
 4 Q. Is Tanacet a natural product?
 5 A. I have no idea. Fever Few.
 6 Q. Do you know whether it's synthetic?
 7 A. I don't know. I don't believe it is synthetic
 8 but --
 9 Q. You say the doses evaluated released two logs
 10 below the Fever Few recommended by DCO, 600 milligrams
 11 to 2,400 milligrams per se?
 12 A. That is Fever Few. I don't know what the
 13 content of parthenolide is in that DCO product.
 14 Q. How did you arrive at the 600 milligrams to
 15 2,400 milligrams a day, 600 to 2,400 milligrams per
 16 day.
 17 A. I had the label and the ones I looked at are
 18 different because I clearly state what the recommended
 19 amounts should be and this one, although I'm having
 20 trouble reading it, I think it says three capsules. I
 21 just can't read the small print.
 22 Q. Are you reading supplemental facts?
 23 A. Supplemental facts, and I'm looking at Fever
 24 Few and I think it says 100 milligrams and that is per
 25 serving and yet a serving is three capsules. You mean

1 produced the labels that were provided within the
 2 course of the investigation. So it's possible they
 3 made some changes subsequent to his report, so I don't
 4 know if it's appropriate to ask him about this.
 5 MR. J. TURNER: We're going to put this in as
 6 an exhibit. This is not a produced document. How can
 7 you ask him questions about something that was not
 8 produced to us? So it's not appropriate to ask
 9 something that is on a subsequent document which
 10 clearly this is.
 11 MR. C. TURNER: How do you know this isn't
 12 the one produced?
 13 MR. J. TURNER: Just wait. We will compare
 14 what you have to this.
 15 MR. PAYNTER: Certainly.
 16 MR. J. TURNER: We got this because we asked
 17 for the thing in color, so it is allegedly to us
 18 identical.
 19 MR. PAYNTER: It would seem it was better to
 20 use the document we Bates stamped, produced in our
 21 production to you. I don't know if we're able to find
 22 that now.
 23 MR. J. TURNER: We'll find it.
 24 MR. PAYNTER: Certainly you're asking
 25 questions, a whole line of questioning based on a label

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1 that is clearly not the label produced in the course of
 2 discovery, which is inappropriate. You can ask him
 3 questions about this new label, but it has nothing to
 4 do with the report.
 5 MR. J. TURNER: We don't know that.
 6 MR. C. TURNER: Off the record for a minute.
 7 (A discussion was held off the record.)
 8 MR. J. TURNER: Withdraw the exhibit.
 9 Q. It's at this point that we have a biomolecular
 10 base that has been discussed above in the next
 11 paragraph and you're saying it was discussed below?
 12 MR. PAYNTER: It was discussed above in the
 13 Bio*Shark.
 14 MR. J. TURNER: Let's go back to that.
 15 A. 16, page 16. It also contains 50 milligrams of
 16 biomolecular base. That's in Bio*Shark.
 17 MR. J. TURNER: Yes. Let's talk about that and
 18 let's make a point that this is a discussion that was
 19 also part of GDU.
 20 Q. With regard to Bio*Shark and GDU there is a
 21 biomolecular base that you refer to. Can you describe
 22 your view with respect to that?
 23 A. Yes. Bio*Shark contains 50 milligrams of
 24 what's called biomolecular base. It contains herbal
 25 ingredients like Eleuthero root, garlic and dandelion.

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1 It also contains elements and minerals, including
 2 barium, bismuth, gallium, silicone, silver, strontium,
 3 titanium, vanadium and zirconium.
 4 I searched the literature, Google and other
 5 sources, to try to determine whether there were any
 6 minimal daily requirements or any essential nutritional
 7 value for any of these elements and minerals and I was
 8 not able to find anything. We use barium for medical
 9 imaging solutions to do a barium enema. We use gallium
 10 in a ray isotropic imaging study for cancer. Silver
 11 I'm not sure what we use that for in nutrition. I have
 12 titanium in my golf clubs and golf balls, but I don't
 13 know whether I need it in my diet. I'm not sure what
 14 the purpose of that is, and I'm not sure what the
 15 nutritional value of any of these things are.
 16 Q. I think we're ready to go on then to BioMixx?
 17 A. Okay.
 18 Q. I have of course the same opening question
 19 about the questions we're focusing on. How did the
 20 questions you're focusing on get formed?
 21 A. Exactly the same way as for the other three
 22 compounds.
 23 Q. You indicate that BioMixx contains a mixture of
 24 so-called biomolecular nutrients. Explain what it is
 25 you're saying there in that part of the report.

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1 A. I'm not sure that biomolecular nutrients is
 2 my -- I originated that or it's in the label of
 3 BioMixx, but it does contain the things I listed here,
 4 goldenseal, echinacea, ginseng, gamma globulin complex,
 5 vitamins, minerals, amino acids and enzymes.
 6 It's got some other interesting ingredients
 7 that merit discussion. It contains guarana, which is
 8 caffeine plus some other things.
 9 It's got a lot of interesting things in it.
 10 One of the interesting things is goldenseal. The DCO
 11 recommended dose -- no. The recommended dose of golden
 12 seal from Cassileth and Lucarelli, and I'm not sure it
 13 comes recommended, it's what is in the available
 14 nutritional sources, is 250 to 500 milligrams three
 15 times a day, which would be 750 to 1,500 milligrams a
 16 day.
 17 Q. Let me ask you a question. When it says
 18 recommended, recommended for what?
 19 A. That's a quote from Cassileth and Lucarelli.
 20 Recommended for -- I have no idea. It is commonly
 21 quoted amounts, some I have no idea, but the important
 22 thing that I talk about is what does goldenseal contain
 23 that might be important from a pharmacological cancer
 24 therapeutic perspective. And the active ingredient in
 25 goldenseal is an alkaloid called Berberine. If you

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1 were to take how much goldenseal is recommended and
 2 what proportion of goldenseal is Berberine, that would
 3 mean a patient might get 4.5 or 90 milligrams a day of
 4 Berberine. If goldenseal was in the product and if
 5 pure goldenseal was taken and if the goldenseal
 6 contained that percentage of Berberine that has been
 7 reported in other goldenseal components -- do we have
 8 the label for BioMixx?
 9 MR. PAYNTER: They don't have labels.
 10 A. We don't have labels.
 11 MR. J. TURNER: Just these we've withdrawn.
 12 A. Because I looked for Berberine as one of the
 13 components of BioMixx, I couldn't find it. So this is
 14 one of the problems I had. There is active ingredient
 15 in something, how much of it is in the product that is
 16 being put forward by DCO and I have no idea.
 17 However, there have been studies of Berberine
 18 in tumor cells in vitro. And you need 50 micrograms
 19 per ML in the test tube to show that it might have a
 20 killing effect on brain tumor cells, either human brain
 21 tumor cells -- this is not in a human with a brain
 22 cancer. It's brain cells, brain tumor cells put in a
 23 test tube in the laboratory to see what concentration
 24 of pure Berberine would kill the tumor cells.
 25 So, again, if you're going to extrapolate from

1 in vitro non-clinical animal studies or petri dish
2 studies and then jump to I'm going to give a patient
3 goldenseal, you have to know how much Berberine is in
4 it, how much of the Berberine gets absorbed and you
5 have to know what levels of Berberine might be in what
6 a patient is getting and do they reach the levels that
7 would be an inhibitor of tumor cell growth, at least in
8 the animal model.

9 Those are the kind of data that you need to be
10 reliable and competent to say this agent has anticancer
11 activity in humans. We don't have that. We don't have
12 any clinical studies of goldenseal. We don't know
13 whether BioMixx contains goldenseal to be active in the
14 animal model, so we can't make any conclusions about
15 Berberine, goldenseal as an active anticancer agent.

16 Echinacea is present in BioMixx. There is a
17 recommendation of five scoops per day, and according to
18 my calculations that would be 25 milligrams of
19 echinacea. Recommended daily doses, whatever they are,
20 would be much much higher than that. 500 to a thousand
21 milligrams, three times a day or about 1,500 to
22 3,000 milligrams of echinacea for other nutritional
23 treatment, as I say, that is echinacea may be helpful.

24 What is in BioMixx is two percent of what is
25 the, quote, daily dose, so it is well under what is

1 recommended.

2 Q. Again, let me ask you, recommended for what?

3 A. Whatever nutritional sources recommend these
4 things. It's not like the recommended daily dose --
5 recommended dose of vincristine to treat acute
6 lymphoblastic leukemia is 1.5 milligrams per meter
7 squared per week intravenously. That is the
8 recommended dose. Every drug label has a dose. Be
9 very careful you are supposed to prescribe to that dose
10 based upon phase I testing, maximum tolerated dose,
11 does limit of toxicity.

12 So we don't know what the recommended dose is
13 for treating cancer patients. It's never been
14 established.

15 Other ingredients here we talked about before,
16 ginseng, bromelain, boron, but then I think there is
17 some novel ingredients that I think warrant discussion.
18 ATP is a high energy phosphate.

19 Q. What is ATP?

20 A. When the body is metabolizing glucose in a
21 process called glycolysis, which is a process which
22 converts glucose to high energy ATP. I have no idea
23 whether 153 milligrams of ATP taken by mouth is ever
24 going to get absorbed. It will still be ATP by the
25 time it gets across the intestinal track, and I see no

1 benefit at all of giving someone ATP if they're having
2 glucose in the diet where they make all the ATP they
3 need, enzymes that convert glucose to lactic acid, and
4 during the process a number of ATP are made in every
5 cell in the body. Taking ATP by mouth is no good. No
6 benefit. It may be of no harm but there's no use of
7 ATP taken this way.

8 Q. When you say no good --

9 A. It's of no use to you. You get ATP not by
10 taking it by mouth. It's not a nutritional supplement.
11 Your body makes ATP unless you have no enzymes to
12 convert glucose to lactic acid. If that were the case,
13 you would be dead. You can take another higher source
14 of ATP, by the way, would be to catch fireflies on some
15 August night and clip off the tail and have tons of ATP
16 because that is where the biochemical companies get the
17 ATP for biochemical reactions that you might do in the
18 laboratory. But that's in a test tube.

19 Q. They get it from fireflies?

20 A. Fireflies. That's why it lights up. It's a
21 high energy phosphate source and lights up at night
22 because it is the ATP. DNA, what use is that? How is
23 that going to help somebody, 1,400 milligrams of DNA,
24 2,900 milligrams of RNA? What kind of RNA is it I ask.
25 Is it viral RNA, is it messenger RNA? What about the

1 DNA? Do you have to take DNA by mouth? If you have
2 meat in your diet, you're going to have DNA. Again, I
3 don't understand the purpose of adding DNA to a diet if
4 somebody is getting protein. And if they're not
5 getting enough protein, there are better ways to get
6 these ingredients than by taking some purified DNA or
7 whatever.

8 The guarana is basically caffeine. It's a
9 stimulant, we all know that. We don't know whether it
10 has any anticancer activity. There is bee pollen in
11 here. There's nothing on the label that I could see
12 that alerted patients to avoid it if they're allergic
13 to bee stings.

14 Q. What is the relationship between bee stings and
15 bee pollen?

16 A. From Cassileth and Lucarelli, there may be
17 allergic reactions to bee pollen for people who are
18 allergic to it.

19 Q. To bee stings?

20 A. If they're allergic to bee stings and take bee
21 pollen, they might have an allergic reaction. It's a
22 risk.

23 Q. The label says BioMixx is used to assist the
24 body in fighting cancer and healing the destructive --
25 that's their quote.

1 A. I could find nothing to support that and the
 2 only way you could do it would be study design that
 3 I've offered them or offered in my report where, again,
 4 it would be a randomized placebo control trial in which
 5 patients who are on the same chemotherapy that may have
 6 adverse, quote, destructive, unquote, effects would get
 7 the chemotherapy with known side effects or radiation
 8 therapy with the same dose, with or without BioMixa or
 9 placebo.

10 Then what I would look at would be given the
 11 doses that DCO is recommending, make sure the patients
 12 have the same disease, getting the same chemotherapy
 13 that has the same adverse effects or the same dose of
 14 radiation over the same period of time for the same
 15 disease and see whether or not BioMixa has a beneficial
 16 effect in decreasing these, quote, destructive effects
 17 of radiation in chemotherapy.

18 Q. What would a study like that cost?

19 A. Depends on how big a study you would want to do
 20 and if it were a phase II study, you might be able to
 21 do it with 40, 50 patients minimally, maybe more. But
 22 let's say 40 patients in each arm of the study where
 23 you would know there would be a certain proportion of
 24 patients who would have side effects of the
 25 chemotherapy or side effects of the radiation, similar

1 severity of all the expectant side effects of treatment
 2 and grade them, mouth ulceration, how severe the anemia
 3 would be, you want to be able see the frequency of the
 4 side effects are different in the patients given
 5 BioMixa or placebo.

6 So it might take three months to complete the
 7 study. Then you analyze all your data. You're still
 8 looking for a number.

9 Q. Uh-hum.

10 A. If you turn it all over to a CRO, leaving out
 11 the cost of the product, which would be provided by the
 12 company and ask them to do everything, probably
 13 \$2 million.

14 Q. That would be a phase II study?

15 A. This would be a phase II study.

16 Q. You think that would be enough to find the
 17 answers you're looking for?

18 A. Certainly give you important information, yes.

19 Q. Now, up until now I thought you needed to have
 20 a phase III study in order to be able to actually come
 21 to a conclusion.

22 A. Depends how robust the data are to show
 23 differences. If you saw a huge value that BioMixa
 24 lowered severity, P value of .0001 compared to placebo,
 25 and this is an important need that cancer patients

1 across all patients and see whether you can decrease
 2 the intensity and severity of those side effects and
 3 they're all measurable.

4 Q. What would that cost?

5 A. Depends on how much help and support the
 6 sponsor wanted in performing the study. How many of
 7 their own resources would they use or if they didn't
 8 have it, they would have to rely on an outside
 9 organization, like a contract research organization, to
 10 manage the clinical trial for them. They would provide
 11 the BioMixa, since these are standard regimens, they
 12 wouldn't have to provide chemotherapy. Radiation
 13 therapy would be standard and you wouldn't have to pay
 14 for that. They would have to provide the BioMixa and
 15 placebo but the contract research organization would
 16 identify the centers, sites or doctors who would
 17 participate in the study. There could be somebody in
 18 practice in Ridgewood, New Jersey in a community
 19 hospital, doesn't have to be a big cancer center. You
 20 would identify the sites, write the protocol, you would
 21 have to write the informed consent, get all the
 22 regulatory documents in order so it could be approved
 23 by the institutional review board.

24 Then you would, since this is mostly a toxicity
 25 study, you would have to record the frequency and

1 have, reduce the side effects of chemotherapy and the
 2 data were very robust and you did a placebo controlled
 3 randomized trial, approval is sometimes granted for
 4 that if it's well designed, carefully controlled.

5 Q. On a phase II?

6 A. Yes.

7 Q. How frequently does a phase II trial lead to
 8 approval?

9 A. Infrequently, but it can happen, particularly
 10 if it's an unmedical need. What the FDA may require
 11 is -- they might grant provisional approval based
 12 upon --

13 Q. Most likely you're saying frequently it's a
 14 phase III study?

15 A. But not always.

16 Q. What would that cost?

17 A. The larger -- and, again, it would depend on
 18 how much the sponsor wanted the organization to cover,
 19 if it was everything, small organization, they wouldn't
 20 have the ability to do the data analysis, monitoring,
 21 site management, review of all the data, writing of the
 22 reports, it might be double that amount.

23 Q. So for the phase III it may be four million?

24 A. If it's twice as many patients and twice as
 25 many sites, yes.

1 Q. And so a phase II and a phase III would be
 2 \$6 million?
 3 A. Well, we said the phase II would be two
 4 million, double it for the phase III.
 5 Q. So phase III would be equal to?
 6 A. Again, I need to have all this reviewed by a
 7 biostatistician to set up what differences we're
 8 looking for and make sure we have adequate numbers of
 9 patients to show differences.
 10 Q. In order to accomplish what you're saying do
 11 you need to do a phase I study?
 12 A. There's so many different things in BioMixx to
 13 do a phase I study with 70 different ingredients you
 14 would hate to do that. How do you do it for this
 15 compound which is so complex? It is not a single
 16 compound. You got tons of different amino acids and
 17 all these other things in here. For some of these
 18 supplementary medical things, like in the shark
 19 cartilage study, we didn't do pharmacokinetics,
 20 pharmacodynamics. What you're looking for is decrease
 21 in toxicity here.
 22 So one could do a very small phase I study to
 23 just make sure that certain ingredients could be
 24 measured and absorbed and it was an acceptable safety
 25 profile.

1 do that with supplemental agents that are attempting to
 2 decrease some of the side effects of therapy.
 3 Q. What is the nature of the difficulty?
 4 A. The complexity of the compound that you're
 5 looking at. It's not a single compound.
 6 Q. So the complexity of the compound makes the
 7 price go down?
 8 A. Well, if it's possible to measure all of the
 9 different ingredients of BioMixx to see what is being
 10 absorbed and what the pharmacokinetics are, that would
 11 be extremely expensive if you wanted to measure all
 12 these things. If you were looking at a single
 13 ingredient, you wanted to look at Berberine in the
 14 goldenseal, you want to pick one ingredient here that
 15 you thought was really going to have anticancer
 16 activity, that would be easier.
 17 If you want to study everything that you claim
 18 is active in BioMixx so you can fill it with all the
 19 different things in it, you would have to measure these
 20 things to see if they're absorbed, how they're excreted
 21 and whether they're having any effect at all on the
 22 other chemo drugs you're giving. That is very
 23 expensive. You're measuring 18 different amino acids.
 24 Once you start getting to that, there is a huge amount
 25 of data that you have to collect to show. That's what

1 Q. What would that cost?
 2 A. A lot less. It is a small study. Might be 36
 3 patients in that study, so much smaller.
 4 Q. How many were in your phase II and phase III?
 5 A. Phase II could be 40 to 80.
 6 Q. 40 to 80?
 7 A. That small. 40 is small, 80 is more
 8 reasonable. Randomized trial might be a couple
 9 hundred.
 10 Q. When you say a couple million dollars, were you
 11 talking about a 40, or 80?
 12 A. The more patients you have is the more money.
 13 Q. I'm asking --
 14 A. I'm giving you numbers that are not my primary
 15 responsibility. I never do the costing of studies.
 16 I'm thinking of similar type of trials that we've done
 17 that are in that range.
 18 Q. But earlier you said that going from scratch to
 19 the completion of a phase III study was about a hundred
 20 million dollars?
 21 A. That was because of the types of agents that
 22 were being developed, early development stages of that
 23 study. The fact that they were anticancer agents, that
 24 would have to be tested very carefully. There are more
 25 pharmacodynamic studies done. It's more difficult to

1 makes these studies very difficult.
 2 Q. So you said you could keep these in your
 3 BioMixx?
 4 A. You're saying BioMixx is important because it
 5 contains all these things, you better measure them.
 6 When we did our shark cartilage study, the only
 7 medicine that patients were getting was the shark
 8 cartilage and the FDA did not ask us to do a PK study
 9 to measure the active peptides that are in shark
 10 cartilage. However, if you're going to give it with
 11 chemotherapy, very often the FDA will ask you to do PK
 12 to make sure it's not having a negative or positive
 13 effect on the basic treatment.
 14 Q. How do you measure the interaction between the
 15 various single entities, synergy?
 16 A. You could be infinity, couldn't it? That's
 17 what makes it complex. It's very difficult to do that.
 18 Q. We discussed tumeric and you talked about one
 19 ingredient, which was a fairly substantial undertaking.
 20 A. Yes.
 21 Q. Do you know how many single entity ingredients
 22 there are in tumeric?
 23 A. The one that seems to be interesting that
 24 everyone studied is curcumin.
 25 Q. That's the one?

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1 A. Yes.
 2 Q. There's about 500 ingredients, so we have the
 3 same problem with working with that?
 4 A. I haven't seen studies to the extent that I've
 5 seen studies on curcumin in cancer, and so if I were to
 6 take the active ingredient, ingredient that is most
 7 promising in terms of its activity, I would look at
 8 curcumin.
 9 Q. What is the underlying theoretical reason for
 10 taking a complete substance made up of 500 units, 500
 11 single chemical entities like tumeric and taking one of
 12 them out and looking at it? What is the rationale for
 13 that?
 14 A. If you start off in the non-clinical studies to
 15 see whether purified active ingredients, any one of
 16 those 500 shows some evidence of anticancer activity,
 17 that would be the way we start.
 18 Q. Why would you do that?
 19 A. There has to be some starting place somewhere
 20 that just chemical or this component has some kind of
 21 anticancer activity, if that is where you want to use
 22 it.
 23 Q. Tumeric has been used in Chinese medicine, you
 24 said in here, for how long?
 25 A. A long time.

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1 Q. And for what purposes?
 2 A. Many purposes, including treating the number of
 3 ailments, including cancer.
 4 Q. So 2, 3,000 years?
 5 A. Does that prove it's an active --
 6 Q. I'm just asking. You're saying we got this
 7 thing, 2 or 3,000 years people have been using for this
 8 purpose, and what we should do is break it down into
 9 500 components and start looking at each one of them.
 10 A. No. I'm saying taking the most active
 11 ingredient, curcumin, and look at it.
 12 Q. How do you know that curcumin is the most
 13 promising?
 14 A. Read the literature and see what has been
 15 looked at.
 16 Q. So when we talked about there being 5,000
 17 promising single chemical entities of which one makes
 18 it all the way through, that's 4,995, and five makes
 19 it, how did the person -- how did the first person that
 20 picked one of the processing entities in tumeric know
 21 which one to pick?
 22 MR. PAYNTER: Objection. That question --
 23 could you --
 24 Q. How did the person that picked curcumin know
 25 they should pick curcumin?

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1 MR. PAYNTER: Objection. How would he know
 2 that? The studies speak for themselves as to why they
 3 were pursued.
 4 Q. Let me ask this question. You got 5,000 items
 5 that you said were promising entities.
 6 A. Yes.
 7 MR. PAYNTER: Okay. That's just pulling out
 8 of the blue. Are you talking about earlier --
 9 MR. J. TURNER: In his report.
 10 MR. PAYNTER: Please reference something.
 11 MR. J. TURNER: In his report he said --
 12 MR. PAYNTER: Please reference what you are
 13 talking about.
 14 Q. Did you understand my question?
 15 A. No, not really.
 16 Q. In your report you say of 5,000 processing
 17 entities that are accumulated, five of them will make
 18 it beyond the initial stage of being looked at and one
 19 of them will make it all the way through the process.
 20 A. Yes.
 21 Q. That leaves 4,995 --
 22 A. Right.
 23 Q. -- that get brushed aside?
 24 A. Right.
 25 Q. On what basis do you know how to pick the one

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1 you're going to study?
 2 A. It's been done since we've been developing
 3 anticancer drugs and that is you do screening and you
 4 do screening by taking purified compounds and you
 5 incubate them with tumor cells and you see whether you
 6 get tumor cell kill or you slow down the rate of
 7 division of the cancer cells, and that's how these
 8 agents screened, and you might find in that 5,010 that
 9 are promising and you move it along to the next stage
 10 of development.
 11 Q. How does that process detect any synergy
 12 between any of the substances in one product?
 13 A. Then you got to do studies of synergy or
 14 additive or negative effects to see that.
 15 Q. And --
 16 A. That's why, you know, these complex compounds
 17 are very difficult to show that they're active because
 18 they're so complex. You never know which is the active
 19 ingredient, but I will go back to my statement, look at
 20 all of the published data on what is in tumeric that
 21 appears to be active. And we're now into clinical
 22 trials with curcumin and not one of the other 490,099
 23 agents that you would like to study or may or not have
 24 been studied, I don't know.
 25 Q. The opening question of this whole line which

1 is what I'm trying to get at is: What is the rationale
2 for taking tumeric, a substance that has been
3 5,000 years or 3,000 years in Chinese medicine, and
4 saying let's break it down into 5,000 or 500 components
5 and look at one of them, what is the rationale for
6 that? Why does that make sense?

7 A. Because it may give you the opportunity to
8 identify the most active agent, avoids the ease of
9 other things that are inactive or may potentially be
10 harmful.

11 Thirdly, just because something has been used
12 for 5,000 years doesn't prove that it's effective and
13 safe in treating cancer patients.

14 Q. Is there any other way to approach it?

15 A. I talked about the process of developing cancer
16 drugs that will indicate whether they're safe and
17 effective in treating cancer.

18 Q. I'm saying is there any other way to do this
19 except the way you describe?

20 A. Not that I know of. Not if you're going to
21 make a claim that this is effective in stopping human
22 cancer growth, curing cancer or preventing cancer.

23 Q. Okay. You mentioned that this was not -- there
24 was no reason to think of this as a food additive. I
25 think it's ATP you were talking about.

1 A. Yes.

2 Q. We've been discussing drugs, foods, dietary
3 supplements. What is a food additive?

4 A. Could be coloring agent, artificial flavor.
5 That is what I look at as additives. I'm not sure how
6 you -- how you're looking at that word "additive."

7 Q. Go back to the question. I'm asking you how
8 the concept of food additives has no function as a food
9 additive that found its way into your discussion of
10 ATP. This is the only place it appears.

11 A. I guess what I meant there is this is a dietary
12 supplement. Food additive means dietary supplement,
13 something you should add to your daily intake of food
14 and it will help you. It's a supplement to your diet.

15 Q. Okay.

16 A. Added to the foods you're already taking is the
17 way I would respond to that.

18 Q. I'm trying to find the reference to Buffalo
19 wings.

20 MR. PAYNTER: Right after ATP.

21 Q. What were you saying there?

22 A. There's 1,400 milligrams of DNA in BioMixx and
23 where did DNA come from? Does it make any difference?
24 Whose DNA is it? Is it human DNA, grasshoppers, bald
25 eagle DNA, Buffalo wings?

1 MR. PAYNTER: What was the question? I'm
2 sorry.

3 MR. J. TURNER: What was the meaning of the
4 Buffalo wings.

5 Q. Shortly after that in the BioMixx discussion
6 you say the argument is that supposedly hundreds of
7 thousands of patients have been treated with DCO
8 products and claim benefit. Where did that come from?
9 Where did you have -- where did you find the hundreds
10 of thousands of patients?

11 A. Where are you now?

12 Q. It's right after you talk about the Buffalo
13 wings, and then the bee sting, and then it's the next
14 paragraph after the bee sting.

15 A. Okay.

16 Q. "All three received" and then it goes on to
17 "Summary and Conclusions." There's a sentence, second
18 sentence in summary and conclusions.

19 A. Okay.

20 Q. "The argument that supposedly hundreds or
21 thousands of patients have been treated with DCO
22 products," where did you find that argument?

23 A. Hundreds isn't a large number and thousands
24 isn't a large number, and I assume there are an awful
25 lot of people buying DCO products. I don't know the

1 exact number. I couldn't find it anywhere, but I don't
2 think a few hundred patients would keep them in
3 business and a few thousand wouldn't be enough either.

4 I don't know the exact number but just because
5 an X number of people took something doesn't prove its
6 benefit. That is not reliable and not competent
7 evidence to support its use or efficacy in treating a
8 particular disease.

9 Another interesting thing is who are the cancer
10 patients who are most likely to take alternative or
11 complimentary or unproven medicines? They're the
12 sickest, the patients with most advanced disease, their
13 patients who have been through multiple courses of
14 chemotherapy and they're most vulnerable to taking
15 things that may be of no benefit to them. They're the
16 most desperate.

17 Q. What do you base that on?

18 A. Recent publication.

19 Q. What is the publication?

20 A. It was in -- I don't know the exact source. I
21 can provide it to you. It was a peer-reviewed article
22 on who are the population of patients most likely to be
23 taking alternative therapies.

24 Q. So you're going to supply us with that?

25 A. Yes.

1 MR. PAYNTER: Sure.
 2 Q. You're saying that those are the kind of people
 3 that are most likely to take Daniel Chapter One
 4 products?
 5 A. Or other alternative therapies.
 6 Q. But we're talking about Daniel Chapter One.
 7 A. That's right.
 8 Q. It's more likely that those kind of people
 9 would take Daniel Chapter One products rather than say
 10 the members of their Christian ministry?
 11 MR. PAYNTER: Objection.
 12 A. I don't know who they are, I'm sorry.
 13 MR. J. TURNER: Objection on what grounds?
 14 MR. PAYNTER: No foundation. How does he know
 15 who buys the products?
 16 Q. You're saying you have no idea who buys DCO
 17 products?
 18 A. No, I'm saying --
 19 Q. You don't know whether the statement made in
 20 that article you're going to give us applies to Daniel
 21 Chapter One or not?
 22 You have to say the words. You can't shake
 23 your head.
 24 A. Yes.
 25 Q. I forgot to tell you that at the beginning.

1 A. Yes, there was a publication in --
 2 Q. You're saying that the position is that
 3 curcumin harms people?
 4 A. I'm saying that anything you take may have side
 5 effects. The idea that herbal medications have no side
 6 effects and chemo radiation just kills people is not
 7 honest.
 8 Q. Do you think that herbs have the same level of
 9 potential negative effects as pharmaceutical drugs?
 10 A. All pharmaceutical drugs, you're combining
 11 every single drug.
 12 Q. Let's deal with cancer treating agents.
 13 A. I can think of a lot of cancer treating agents
 14 that don't have a lot of side effects.
 15 Q. Can you tell me some that don't --
 16 MR. PAYNTER: Can you allow him to finish
 17 answers before you jump in?
 18 A. There are many classes of anticancer agents.
 19 Some are what we call cytotoxic agents, classical
 20 chemotherapeutic agents that kill cancer cells but they
 21 also can damage normal cells. Commonly the use of
 22 chemotherapeutic agents used in treating leukemia are
 23 beneficial but have side effects.
 24 A newer class of anticancer agents are more
 25 specific of what they're going after in the cancer

1 A. The article doesn't go into patients who might
 2 be taking or not taking DCO products. It is just who
 3 are the patients with cancer most likely to take
 4 alternative therapies or unproven therapies. I don't
 5 have an idea whether people who take DCO products are
 6 different from the population.
 7 Q. When you say "unproven" is that the same as
 8 disproved?
 9 A. Unproven means there's been no reliable or
 10 competent evidence to support the efficacy or safety of
 11 that particular product in treating a cancer patient.
 12 Q. Are there safety issues about the DCO products
 13 you reviewed?
 14 A. In some patients maybe.
 15 Q. What do you mean by that?
 16 A. Some of the products may interfere with the
 17 activity of certain chemotherapeutic agents.
 18 Curcumin -- and I alluded to curcumin more than I have
 19 other drugs or agents. A recent study was just
 20 published in January of this year that indicates that
 21 curcumin combined with iron and patients who have
 22 chronic disease like cancer, they become iron deficient
 23 and it's possible anemia caused by a revoke and restore
 24 deficiency would worsen.
 25 Q. Has that been established?

1 cell. So because they're much more specific and
 2 because these are targeted therapies, we find that side
 3 effects are much less than the classical cytotoxic
 4 agents.
 5 Q. Do you think in general herbs have the same
 6 level of side effect as the old class of drugs?
 7 A. I found that very effective anticancer agents
 8 often will have side effects and that the idea that
 9 there's something out there that is active in treating
 10 cancer and has no side effects at all I think is a
 11 figment of imagination. It doesn't happen.
 12 Q. So you're saying herbs that might effect cancer
 13 and the older category of drugs that might effect
 14 cancer both have side effects?
 15 MR. PAYNTER: He never said anything about
 16 herbs that effect cancer. You're reading into his
 17 testimony. He never testified there are herbs --
 18 Q. You don't believe there are any herbs that
 19 effect cancer?
 20 A. I don't know of one herb -- I'm going to
 21 exclude plant derived chemotherapeutic agents. There
 22 are a number of agents that are cytotoxic that
 23 originally came from plants or the bark of the yew tree
 24 that are now made synthetically, vincristine came from
 25 a plant. The taxane, paclitaxel, came from the yew

1 tree. Now it's made synthetically. There are plant
2 derived cytotoxic agents and a lot of medicines came
3 from plants. I will not deny that.

4 I will state I'm unaware of any of the herbs or
5 ingredients of any DCO product that has been shown with
6 competent and reliable evidence in patients with cancer
7 that they have a beneficial effect in decreasing growth
8 of tumors, curing tumors or preventing tumors.

9 Q. And do you have any credible scientific
10 evidence that they don't?

11 A. You have to tell me that they do. You have to
12 show me they do.

13 Q. You have proven that these products don't have
14 any --

15 A. You have to show me. You're saying they are
16 going to be used to treat somebody's cancer or decrease
17 the destructive effects of cancer therapy and to say
18 that you have to do the studies to do it.

19 Q. Is this a legal conclusion?

20 A. Medical conclusion, scientific.

21 Q. So it's not a legal conclusion?

22 A. I'm not here to make legal conclusions. I'm
23 here to give you scientific evidence of what is valid
24 and isn't. I devoted my whole life to helping kids and
25 now adults in fighting cancer to diminish the side

1 tried to reduce the doses of chemotherapy they were
2 getting, so they weren't getting so sick, they still
3 got sick. Something interesting is going on here.

4 And we determined that this young patient whose
5 mother was ready to stop her chemotherapy, very highly
6 educated woman whose parents were physicians, father
7 was a pathologist, she was a teacher, we decided to
8 look at it her way. She metabolized chemotherapeutic
9 drugs. It turned out she inherited from her mother and
10 father a gene that decreased the ability of the patient
11 to actually detoxify that chemotherapeutic drug. There
12 was a defect in the enzyme that metabolized it.

13 We wound up -- I sent blood samples on the
14 mother, father and child to St. Jude's Hospital. This
15 case has been published. And they found she was
16 lacking the enzyme, and her parents were both carriers
17 of the enzyme deficiency and we reduced her dose of one
18 of the chemotherapeutic agents from 50 milligrams a day
19 to 12.5 milligrams a week. That is a huge reduction.
20 Because that 12.5 milligrams a week was enough to keep
21 her disease in remission and she remained in remission,
22 she had no more side effects and she's now back -- this
23 was back when I was at Cancer Treatment Centers of
24 America. That was back in the '90s. She's cured.

25 What I'm trying to say is the more we learn

1 effects of treatment and prolong their lives and done
2 it at a very rigorous, difficult, not easy way. It's
3 been very, very arduous but the end results are better.

4 Today 80 percent of children with leukemia are
5 being cured. When I first started in this profession
6 of mine virtually every patient died.

7 Q. What percentage of adults with leukemia are
8 cured?

9 A. What kind of --

10 Q. The one you just used for children.

11 A. Acute lymphoblastic for adults are not as good.
12 Acute myeloid leukemia are not even as good. There are
13 other ways to treat those patients. If you can induce
14 a remission in a patient with acute myeloid leukemia,
15 adult patient or acute lymphoid leukemia and they have
16 a relative that is a match, they can be treated with a
17 stem cell transplantation and they can be cured.

18 Q. Okay.

19 A. I don't know of a patient of mine who had
20 leukemia who is cured with any herbal medication. I've
21 had patients who were very upset or got very sick from
22 the toxic effects of chemotherapy and went off to
23 Mexico or went down to the Caribbean for unproven
24 therapies and they came back, and I saw them in
25 consultation. And the interesting thing was when I

1 about cancer, what causes it, the biochemical pathways
2 that are involved in cancer development, better ways to
3 attack those pathways with specific drugs that have a
4 known mechanism of action and non-toxicity, we're going
5 to continue to make advances. There are no shortcuts
6 in curing cancer. There is not a shortcut. You try to
7 take the short cut, you're going to wind up with either
8 unexpected adverse effects or ineffective therapy, and
9 I don't think we should do that to patients.

10 (A recess was taken.)

11 Q. You mentioned taxol.

12 A. Yes.

13 Q. Where was taxol discovered? You said a plant?

14 A. No. The yew tree. I think came from China.

15 Q. Yew tree?

16 A. Y-E-W, the bark of it.

17 Q. And what was the process for it to be
18 developed, do you know how?

19 A. I don't know the full history of that.

20 Q. Okay. What, if any, is the value of
21 traditional uses of these herb products that we've been
22 discussing, the traditional use, any value to that?

23 A. I'm not sure I understand what you mean by the
24 traditional use. In what disease? What entity?

25 Q. Let's take -- you mentioned Chinese medicine in

1 your report and a lot of products, a lot of herbs have
 2 been used in Chinese medicine.
 3 A. Yes.
 4 Q. And a lot of knowledge has been attributed to
 5 that use.
 6 A. Yes.
 7 Q. What value is that to us in the present medical
 8 situation about cancer?
 9 A. I think from following lower and common usage
 10 some may come up with some leads that warrant further
 11 development. Following lower and common usage doesn't
 12 prove that something is active, safe and effective but
 13 it may provide leads for further investigation, further
 14 experimentation, further discovery.
 15 Q. Is there any current cancer drug that is
 16 100 percent effective?
 17 A. No. 100 percent effective, that cures all
 18 cancer?
 19 Q. No. For any cancer, cures all people with
 20 cancer X.
 21 A. I'm unaware of any cancer that is curable in
 22 100 percent of the cases that are cured by a drug. I
 23 can think of a number of cancers that can be cured by
 24 surgery, like melanoma if it's diagnosed early, or
 25 basal cell carcinoma of the skin, certain cervical

1 with liver cancer. There are many known causes of
 2 cancer, but there are a lot of cancers we don't yet
 3 know what the cause is. If you were to ask me what
 4 causes childhood lymphoblastic leukemia, I don't know
 5 yet. It's interesting because we can cure it but we
 6 don't know the cause.
 7 Q. In your career do you know how the incidents of
 8 the childhood cancers has grown or diminished?
 9 A. It varies depending on the different type of
 10 cancer.
 11 Q. What is the one that has had the least amount
 12 of increase or the most amount of decrease?
 13 A. I have to look that up. I'm a little tired
 14 right now.
 15 Q. Okay. How about do you know which ones are the
 16 most, increased the most?
 17 A. I think the lymphomas are the group of cancers
 18 that increased a lot in the pediatric population.
 19 Q. When you say "a lot" --
 20 A. I don't know the exact percentage.
 21 Q. Would it be 50 percent?
 22 A. No.
 23 Q. Ten percent?
 24 A. Probably less than that.
 25 Q. Less than ten?

1 cancers.
 2 Q. These are all by surgery?
 3 A. Mostly by surgery or radiation, or could be by
 4 cryotherapy. If it's small and early stage, it can be
 5 excised completely surgically and cured. If you had a
 6 choice between using surgery in an early stage melanoma
 7 or chemotherapy, I would hope that everybody would pick
 8 surgery.
 9 Q. Say that again?
 10 A. If you had a chance of treating early stage
 11 malignant melanoma with surgery or chemotherapy that
 12 might be used for later stage disease, I would hope you
 13 would certainly use surgery. It's much more effective.
 14 Q. Okay. What causes cancer?
 15 A. There are many different causes of cancer,
 16 inherited gene defects inherited from one generation to
 17 the other. There are other causes from external
 18 agents, like viruses that can cause cervical cancer or
 19 hepatitis virus that can go on and increase the risk of
 20 liver cancer. Radiation therapy or radiation itself
 21 can cause cancer. Other chemicals, like benzene, can
 22 cause leukemia. There are genetic factors,
 23 environmental factors, lifestyle factors. We certainly
 24 know the carcinogens in tobacco smoke can cause cancer.
 25 We know that alcohol can damage the liver and result

1 A. We're not talking about big increases.
 2 Q. So pretty much stayed steady?
 3 A. Well, there's been a slight increase in some
 4 and plateau in others.
 5 Q. How about for adult cancers?
 6 A. It's interesting. We're seeing an increase,
 7 for example, in non-small cell lung cancer in women and
 8 a plateau or decrease in men. We've seen a decrease in
 9 stomach cancer in both sexes, I think because of the
 10 concerns about dietary things. We've seen an increase
 11 in lymphoma in adults that may be environmental,
 12 occupational. We're going to see a decrease in
 13 mesothelioma related to asbestos.
 14 Q. Dying off?
 15 A. Protected in the workplace now so they're not
 16 exposed as much. It takes 40 years. We see a decrease
 17 in some. I think with preventive medicine I think we
 18 can see a decrease in many other tumor types or
 19 diagnosis them earlier, colorectal cancer diagnosed at
 20 an earlier stage. Breast cancer has an excellent
 21 prognosis diagnosed at an early stage. We need cancer
 22 preventive changes, overweight, decreased exercise have
 23 a negative effect on one's risk of developing cancer.
 24 How many cancers could we obviate if we stopped
 25 smoking, outlaw tobacco. Alcohol is a major problem in

