

OPEXA THERAPEUTICS, INC.

FORM FWP

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Following is a transcript of a physician question and answer video regarding
Tovaxin made available by Opexa Therapeutics, Inc.:

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TRANSCRIPTION OF DVD

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1 QUESTION: What is multiple sclerosis?

2 DR. EDWARD FOX: Multiple sclerosis is
3 an autoimmune disease, and like other autoimmune
4 diseases there are specific targets that are
5 involved.

6 In this case with multiple sclerosis,
7 the target is myelin that's on the surface of the
8 nerve cells, the axons or the connecting wires that
9 are within the brain and the spinal cord.

10 And so the symptoms of multiple
11 sclerosis, whether it be weakness or numbness,
12 coordination problems, vision problems or many other
13 symptoms that can occur, may occur either
14 sporadically, coming and going, or can be a
15 continuous course.

16 So multiple sclerosis is not truly one
17 disease. There are several types of MS.

18 What's being looked for in this clinical
19 trial are those patients who have relapsing-remitting
20 disease. And that means they'll have a flareup of
21 symptoms and then that flareup may or may not improve
22 over time, and that's called a remission if there is
23 improvement.

24 Relapsing-remitting multiple sclerosis
25 begins with an individual flareup. There is a

1 specific event that may be called a clinically
2 isolated syndrome. If a patient then goes on to have
3 new or worsening symptoms, then it's clinically
4 definite multiple sclerosis.

5 Patients with primary progressive MS do
6 not ever have these types of relapses. They just
7 have a continuous course from the beginning.

8 Our trial is designed to look at those
9 patients with relapsing-remitting multiple sclerosis
10 in an effort to reduce the number of flareups and
11 thereby prevent disability over the years.

12 QUESTION: What is multiple
13 sclerosis?

14 DR. CLYDE MARKOWITZ: In terms of what
15 is MS, we believe it to be an autoimmune reaction to
16 something, we don't know exactly if it's a virus or
17 some toxin, in genetically susceptible individuals
18 that causes their immune cells to attack their
19 myelin.

20 That myelin gets destroyed. Having the
21 myelin stripped away from the nerve cells leaves
22 those nerves nonfunctional.

23 What that ultimately does for patients
24 is it leaves them with some neurologic problems.
25 They may have loss of vision, they may have sensory

1 complaints, numbness, tingling, they may have
2 weakness, bladder problems, and all of these things
3 eventually lead to increasing amounts of disability
4 over time.

5 The problem with MS is that people may
6 have an attack and get better, and then after
7 repeated attacks they don't completely recover and so
8 they are left with some residual neurologic
9 disability.

10 QUESTION: What is multiple sclerosis?

11 DR. BRIAN LOFTUS: Multiple sclerosis
12 falls into a group of diseases that we call
13 autoimmune diseases. What autoimmune diseases mean
14 is the patient's immune systems, which is, of course,
15 designed to fight invading bacteria, viruses and
16 fungi and other bad things that come into your
17 body, gets confused and starts attacking parts of the
18 body.

19 So in multiple sclerosis, the immune
20 system that we think is principally confused is
21 what's called the T-cells, and those T-cells start to
22 attack the myelin. And myelin is the insulation that
23 is around, like a wire, that connects the different
24 parts of the brain together as well as the brain and
25 the spinal cord.

1 Unlike electricity, when you strip off
2 the insulation off a wire, the wire still works. In
3 the brain, these wires -- once the insulation is
4 stripped away, those wires no longer work. So the
5 wires themselves are called axons and the myelin gets
6 attacked. And the myelin gets attacked not on a
7 constant basis, but in a way that we don't understand
8 on sort of a periodic basis.

9 So, typically, MS will start with what
10 is termed "an attack." "An attack" just means that
11 you have a series of symptoms that suddenly develop
12 kind of out of nowhere. And common first attacks in
13 MS can be blindness, another common attack in MS can
14 be like numbness over part of the body or weakness
15 over part of the body, and everybody can be
16 different.

17 And then after you have your attack, for
18 most patients there's a period of healing and
19 patients commonly recover back to -- back to normal
20 at first. And then, unfortunately, after a series of
21 attacks, there is generally some permanent damage
22 that occurs and over time people get progressively
23 disabled.

24 How many people get disabled versus how
25 many people have a sort of automatically benign

1 disease is actually a very controversial subject, but

2 most people think around 10 to 20 percent of MS

3 patients, even if not treated, will do quite well in

4 their lifetimes, but the majority of MS patients need

5 therapy.

6 QUESTION: How is multiple sclerosis
7 treated?

8 DR. EDWARD FOX: The current medications
9 available that are FDA licensed for MS all work on
10 the immune system in an effort to reduce the
11 inflammatory effects seen in MS.

12 So the medications that are FDA approved
13 right now, by and large, the first-line medicines are
14 injections. There are interferons. There are three
15 different interferons that are FDA approved right
16 now, and there is glatiramer acetate, which is a
17 noninterferon drug.

18 All of these are administered by
19 injections. They can be subcutaneous injections,
20 which are just under the skin, or can be
21 intramuscular injections that are deeper injections.

22 There's a variable timeline for how
23 these medicines are given. They range from once a
24 week up to once a day.

25 On the first-line medications that are

1 FDA licensed right now, the benefits of them include
2 a reduction in the number of new MRI lesions or
3 larger MRI lesions. There's also a reduction in the
4 relapse rate, the frequency at which a patient may
5 have new or worsening symptoms. It has also been
6 seen in longer studies that they do have a benefit in
7 terms of reducing sustained accumulation of
8 disability as well.

9 The current medicines that are licensed
10 to treat relapsing-remitting multiple sclerosis are
11 incomplete in terms of their efficacy. There is not
12 a complete reduction in new MRI activity, there is
13 not a complete reduction in the rate of relapses or
14 prevention in disability.

15 There is a gap between where we are in
16 terms of current treatment and where we would like to
17 be in terms of assuring patients that they are not
18 going to progress over time.

19 And so the unmet need right now that we
20 have right in multiple sclerosis is that we want
21 medicines that are -- that are safe and tolerable but
22 also have a greater efficacy than the current
23 medications.

24 The safety of the first-line
25 medications, glatiramer acetate and the interferons,

1 is fairly good. These have been on the market long
2 enough that we're fairly certain of their safety.

3 The tolerability of the medicines is
4 somewhat limited, however, because they are available
5 in injection form that are generally
6 self-administered at home and fairly frequently
7 administered as well. So the tolerability is
8 something that has always been an issue with the
9 current medications, but the safety has been fairly
10 good.

11 There are newer agents that have been
12 FDA approved. Natalizumab is an intravenous
13 medication that has been approved generally for
14 patients who have had an incomplete response to other
15 medications, and safety has been a greater concern
16 for those medications. The reason is because the
17 stronger a medicine is on the immune system, the more
18 likely it is to suppress the normal immune system and
19 lead to an increased incidence of complications.

20 We have had incomplete ability to keep
21 patients on the current medications because of
22 tolerability issues. Injections cause local
23 reactions and can cause reactions that definitely
24 limit a patient's desire to stay on the medication.

25 We have been working very closely with

1 patients for a number of years to attempt to find a
2 medication that both is effective and tolerable, but
3 we have struggled with that.

4 The current therapies are fairly
5 frequent and ranging between weekly and daily and are
6 usually self-administered by the patient in the home.

7 Any medication which is given less
8 frequently is more apt to cause better compliance or
9 adherence to that therapy, because if a patient is
10 well aware of how many injections are going to be
11 necessary during the course of the treatment and what
12 the follow-up is going to be, I think that a patient
13 can enter into the research protocol or into the
14 treatment plan with expectations of continuing on the
15 therapy.

16 QUESTION: How is multiple sclerosis
17 treated?

18 DR. BRIAN LOFTUS: So the therapies we
19 have are sort of two different arms at this point.

20 We do have the acute treatment attack
21 therapy, which is steroids. So you use a high dose
22 of IV steroids or a high dose of oral steroids in
23 order to limit that first attack or some subsequent
24 attack.

25 And then we have a bunch of therapies

1 that we think alters the natural history of the MS
2 disease, and these therapies are, therefore, called
3 disease-modifying drugs and there are several on the
4 market.

5 The ones that are approved by the FDA
6 are Avonex, Betaseron, Copaxone and Rebif. And those
7 are simply in alphabetical order. I don't have a
8 preference among them.

9 They belong in two groups. So one group
10 is the interferons, and that's the Avonex, Rebif and
11 Betaseron; and then Copaxone is its own group.

12 And then recently a new immunomodulating
13 drug on the market is Tysabri. Tysabri is one of the
14 newer agents available. It's a broad spectrum
15 immunosuppressant that basically prevents the T-cells
16 from getting into the brain as well as some other
17 places on the body, and it's sort of the first of a
18 whole series of new drugs that sort of approach the
19 treatment of MS the same way, and that's to keep part
20 of the immune system out of the brain.

21 The upside is they are very powerful
22 drugs and they work extremely well. The downside is
23 they may cause lots of serious side effects. In
24 fact, Tysabri was pulled off the market for a period
25 of time because of one of these side effects.

1 The final drug that's available now is
2 Novantrone. It is the only agent that is really used
3 for secondary progressive MS, and it does seem to
4 help patients for the couple of years that they can
5 take it. Unfortunately, because of the toxicity of
6 this drug, its use is limited for only about two
7 years of therapy.

8 QUESTION: What are the current
9 treatments and unmet needs?

10 DR. CLYDE MARKOWITZ: Over the last 15
11 or so, maybe even 20 years, we've started to look at
12 compounds that look like they affect the immune
13 system. Not immunosuppressants, but
14 immunomodulators. And these immunomodulators
15 ultimately regulate parts of the immune system in a
16 way that prevents some of the damaging effects from
17 the inflammation that occurs in MS.

18 The Interferon products were the first
19 ones that actually came to market, and we've been
20 using them safely for the last 15 years without any
21 major problems. They reduce the number of lesions
22 that are seen on an MRI scan, they reduce clinical
23 relapses and they slow disability progression.

24 Glatiramer came to market a little bit
25 after the interferons and that also has shown the

1 same effects in terms of slowing the disease and
2 preventing the lesions.

3 So overall I'd say, you know, 15 years
4 into this we have some therapies, but there's a lot
5 of unmet needs.

6 First of all, all of these compounds are
7 injectable therapies. Patients have issues about not
8 wanting to take these self-injectable therapies.
9 They have side effects. You know, there are a
10 variety of issues that have to be managed.

11 And after taking injectable therapies
12 for many years, some of these injections leave people
13 with some dermatologic things. They end up with some
14 loss of the normal skin and they end up with little
15 divots that end up creating cosmetic problems for
16 them.

17 After injecting many years, you get scar
18 tissue below the injection sites, and that makes it
19 more difficult to actually inject and can be more
20 painful.

21 From an efficacy standpoint, you know,
22 the therapies that we currently use are modest in
23 their effects. You know, they reduce the activity on
24 MRI and they reduce clinical relapses, but patients
25 still have attacks and still have new lesions, so

1 over time there's breakthrough disease activity.
2 We as investigators have to look for new
3 compounds that will be easier to tolerate, more
4 effective and, you know, ultimately compliance will
5 be better.

6 QUESTION: What is Tovaxin?

7 DR. EDWARD FOX: The concept of Tovaxin
8 is very different than existing medications because
9 it is tailored to an individual. It is not a
10 medication that is exactly the same one person to the
11 next.

12 When looking at somebody's immune
13 system, which is extraordinarily complicated and
14 quite variable from one person to the other, it's
15 reasonable to assume that the type of cells that are
16 causing MS in one person may not be exactly the type
17 of cells that are causing the disease in another
18 person, and so with the concept of Tovaxin we are
19 hopefully tailoring it to the individual.

20 The way Tovaxin is made is via blood
21 products from the individual patient, and so using
22 those cells as the target for the immune system, the
23 idea is to reduce the cell count that is available
24 within a person of cells that would attack myelin and
25 thereby cause the disease.

1 The potential advantages of
2 immunotherapy such as Tovaxin is that it is tailored
3 to the individual. Because the variability exists in
4 the disease, it may be that targeting specific cells
5 that are causing the pathology of MS will have a
6 better outcome in terms of reducing the pathology of
7 the disease.

8 In the Tovaxin trial currently, the
9 patients are coming into the clinic to get the
10 administration of the medication and they are not
11 self-administering at home.

12 It would be expected, as with all other
13 medicines that are administered within a doctor's
14 office or some other medical facility, that
15 compliance or adherence to the therapy would be
16 easier for both the patient and the physician.

17 In the current trial of Tovaxin, there
18 are five injections that are being given, and
19 certainly we're looking at the tolerability of those
20 five injections.

21 It would be expected, given our
22 knowledge that the current injectable medications are
23 given very frequently, that a less frequently given
24 treatment for MS would be met with a greater ease of
25 administration and hopefully an equal or better

1 tolerability.

2 QUESTION: What is Tovaxin?

3 DR. CLYDE MARKOWITZ: So Tovaxin is an
4 interesting compound because, first of all, it really
5 relies on the patient's own cells in their body to be
6 the treatment, in essence.

7 One of the problems you look at with all
8 of the current therapies that are in development
9 right now is that safety is a concern. A variety of
10 the compounds we're testing right now may have other
11 issues, some of which are that they may develop some
12 autoimmune conditions, there may be some liver
13 toxicity, some of them are immunosuppressant, they
14 may raise risks for infections. So all of those
15 issues of safety are of big concern.

16 Where Tovaxin comes into play is that
17 right now what you're asking the patient's body to do
18 is to generate suppressor cells, and the mechanism of
19 how this drug works is kind of unique.

20 First of all, what's done is that they
21 harvest blood for patients and they first determine
22 whether or not they carry a certain level of
23 reactivity to myelin antigens.

24 If they carry that level of myelin
25 antigen reactivity a larger sample is taken, it is

1 grown up, those cells then get irradiated. Once they
2 get irradiated they no longer divide and they now can
3 serve as a target for the immune system, and so
4 they're given back to that patient and that patient's
5 own cells in their body will go and attack and
6 suppress these myelin-reactive cells.

7 Now, from a theoretical standpoint, it's
8 a very, very powerful way to treat a disease that you
9 don't know exactly which antigen is responsible for
10 this disease. And, you know, when you look at
11 proteins as antigens, certain sequences of the
12 protein will be important for one patient and
13 different for another patient. So if you use the
14 same protein for everybody, you may end up getting
15 some responders and some nonresponders.

16 But if you go ahead and give them a full
17 panel of all of the available proteins, you let that
18 patient's immune cells pick the ones that they're
19 reactive to, they grow up against that and then those
20 are the ones that you're going to use as the, so to
21 speak, target for their own cells to suppress.

22 So what's believed to be happening in MS
23 is that there must be some sort of a regulatory loop
24 that keeps these myelin-reactive cells in check in
25 most people normally. If you lose that suppressor

1 activity, they can grab hold and cause activity and
2 disease, and there have been studies over the years
3 that have shown that MS patients lack some suppressor
4 activity.

5 So, the strategy with Tovaxin is to go
6 ahead and try and boost that suppressor activity, and
7 the way it's done is by saying these are the
8 myelin-reactive cells, these are the ones that we
9 need to get suppressed, we can boost their
10 suppression by giving the patient large quantities of
11 it.

12 But since they can't divide or react
13 because they've been irradiated, you're just
14 stimulating those suppressor cells to develop, they
15 can then go in to the brain and go ahead kill the
16 cells that are normally driving the immune response.

17 So I'm an investigator for the clinical
18 trial that's ongoing right now in a Phase II study
19 and, you know, there have been some initial hurdles
20 that we've had to deal with in terms of harvesting
21 the blood, making sure that, you know, patients get
22 their vaccination, but in reality it's gone pretty
23 smoothly. So I'm hopeful that this will be a
24 therapeutic intervention that will move forward.

25 QUESTION: What is Tovaxin?

1 DR. BRIAN LOFTUS: The company takes the
2 patient's blood and identifies the T-cell clones that
3 are responsible for causing the multiple sclerosis in
4 the patient.

5 These T-cells are then multiplied up and
6 they are then irradiated and made them -- and are
7 made inactive and are then given back to the patients
8 in very large amounts, and then the rest of the
9 immune system recognizes these T-cells as being sort
10 of like an invading T-cell and then the rest of the
11 T-cells then attack these T-cells. So it's thought
12 to be like a T-cell vaccine where you trigger the
13 immune system to then attack these T-cells.

14 The biggest advantage of a product like
15 Tovaxin is that it is very narrowly targeting the bad
16 part of the immune system. So all of our therapies
17 for multiple sclerosis target whole classes of the
18 immune system that are felt to be important in the
19 multiple sclerosis disease, but are probably
20 important in defense against disease as well.

21 In the case of Tovaxin, we are
22 especially targeting those immune cells that are
23 causing the damage with very little relative effect
24 to the rest of the immune system. So the side effect
25 profile from an immune system standpoint should be

1 very good with this kind of therapy.

2 The other advantages would simply be the
3 relatively few number of treatments in a year a
4 patient would need compared to taking the current
5 drugs that require injections from once a week to
6 every day and the ability to potentially put the
7 disease into remission for extended lengths of time.

8 QUESTION: What are the results of the
9 PhaseI/II dosing study?

10 DR. BRIAN LOFTUS: I have been the
11 principal investigators on two studies for Tovaxin.

12 The first of these was a dose-ranging
13 study to determine how much of these T-cells should
14 be given back to the patient as the vaccine. And
15 basically in this study there was three groups, a low
16 group, a mid group and a high group, and there were
17 approximately five patients in each group.

18 And what we did is we injected them with
19 T-cell vaccine at different doses and we measured the
20 number of bad T-cells that we could find. And over
21 the course of a year, it was determined that clearly
22 the mid dose and the higher dose did a better job of
23 eliminating the bad T-cells than the low dose.
24 Technically, it's easier to make the mid dose than
25 the high dose, so the future studies have gone

1 forward using the mid dose.

2 In the dose-escalation study, this group
3 of patients is entering an experimental protocol and
4 are the first ones to receive a vaccine of this type.
5 So if you can put your shoes in the patient's shoes,
6 what kind of patient would want to come into a study
7 like that, and basically it's people who have tried
8 the other treatments that are available and they have
9 not been helped by them.

10 So in order to get into the initial
11 dose-escalation study, you had to have failed
12 multiple other therapies for your multiple sclerosis.
13 Because of that, these patients are relatively far
14 along and most of them were quite disabled.

15 This in contrast to the TERMS Study and
16 to where we would ordinarily think that we would look
17 to use Tovaxin in the future, which is when people
18 are in that early relapsing phase before they become
19 disabled. But having said that, those are the kind
20 of folks that are studied.

21 So what we'd found over the year in all
22 groups was that the T-cell vaccine Tovaxin did
23 accomplish what was expected of it, namely that it
24 eliminated or at least decreased, in the case of the
25 low dose, the bad T-cell lines that we were targeting

1 to be -- to be eliminated by the Tovaxin.

2 So if the theory is correct that these
3 bad T-cells cause the MS attacks, then Tovaxin, by
4 eliminating those bad T-cells, would be thought to
5 prevent those MS attacks. We did find that the
6 number of MS attacks did drop among those patients as
7 well.

8 The study is five patients in each group
9 and, therefore, it's a very small study. Also, to
10 see changes in disability generally take longer than
11 one year, so there was a trend toward some improved
12 disability, but nothing that was statistically
13 significant, but given the small numbers of patients
14 involved, that is not a surprise.

15 QUESTION: What are the results of the
16 Phase I/II retreatment study?

17 DR. BRIAN LOFTUS: The second study,
18 which I was an investigator on, was a study to treat
19 patients who'd previously had another kind of T-cell
20 vaccine. So Tovaxin is referred to as a trivalent
21 vaccine because it has T-cells potentially in three
22 different myelin components. Older T-cell vaccines
23 had only one or two.

24 In this case, we used patients who had

25 had a T-cell vaccine that we would refer to now as a

1 monovalent vaccine who had done well with that
2 vaccine who desired to be vaccinated again, to come
3 in and follow a group of them along over time.

4 The advantage of this was to see if we
5 could show that, even after having had prior T-cell
6 vaccine, would Tovaxin still be helpful for this
7 group of patients.

8 What was demonstrated, again using the
9 mid dose that was determined from the first study, is
10 that we can -- we can eliminate the bad T-cells from
11 patients who had had prior T-cell vaccines as well.

12 The extension study patients were chosen
13 from a pool of patients who had previously received
14 monovalent vaccine and then after they no longer had
15 access to the vaccine has had a recurrence of their
16 MS symptoms.

17 And these patients were successfully
18 treated with Tovaxin and their T-cell counts did go
19 down dramatically. And actually in this group, even
20 though it was a one-year study only, a significant
21 percentage of them actually had improvement in their
22 disabilities course as well. That's somewhat of a
23 surprise given, again, a very small study and a short
24 length of time.

1 QUESTION: What is the TERMS Phase IIb
2 Study?

3 DR. EDWARD FOX: I'm the lead
4 investigator for the TERMS trial, the Phase IIb
5 trial, regarding Tovaxin treatment of early MS. This
6 is a trial that is investigating whether Tovaxin is a
7 clinical benefit to those patients who have been
8 newly diagnosed with multiple sclerosis or have had a
9 clinically isolated syndrome where they have very
10 high risk of converting to clinically definite
11 multiple sclerosis.

12 The design of the trial was to
13 investigate whether these patients would benefit,
14 first of all, in terms of reduction on new lesions
15 seen on MRI of the brain. And so the design of this
16 trial is a double-blind, placebo-controlled trial
17 where neither the patients nor I know whether the
18 Tovaxin or a placebo is being administered as an
19 injection.

20 We're following these patients with
21 serial MRIs and the primary end point that we're
22 looking for is reduction in development of new
23 enhancing lesions seen on MRI for the patients who
24 are receiving Tovaxin when compared to those patients
25 receiving the placebo.

1 In a Phase IIb trial that is designed to
2 be a fairly short-term, maybe one year in this trial,
3 other trials go on as long as two years, you're
4 looking for a surrogate marker for disease activity.
5 You're looking for a way to see how a patient is
6 doing without necessarily having to wait until they
7 have sustained accumulation of disability.

8 There have been a number of trials
9 looking at MRI markers as a measure of disease
10 activity and it certainly has been found that the
11 more enhancing lesions a patient has early in the
12 course of the disease, the more likely they are to
13 have another relapse or to have a clinical change
14 early in the course of the disease. So at this stage
15 what we're looking for in the Phase IIb trial is a
16 reduction in new MRI activity.

17 The earliest stages of any trial are
18 usually to determine the appropriate dosing of an
19 agent that's being used and also be able to look
20 preliminarily at markers that can be used to follow
21 patients to determine whether they're having a
22 clinical benefit.

23 And so the earlier trials that were done
24 on Tovaxin were used to help design this trial to
25 determine when the injections should be performed and

1 when the MRI should be performed in order to see a
2 clinical benefit.

3 Safety issues are always a concern in
4 any clinical trial, so that is being monitored
5 carefully in this trial and will be monitored as well
6 in the future. It is unknown right now whether the
7 safety profile will be better for a specified
8 immunotherapy such as Tovaxin, but it's certainly our
9 hope that we are going to target a therapy that is
10 extremely well tolerated and safe as well as
11 effective.

12

13 QUESTION: What is the TERMS Phase IIb
14 Study?

15 DR. BRIAN LOFTUS: The current TERMS
16 Study that's being run by Dr. Ed Fox is really going
17 to prove or disprove the entire concept behind
18 Tovaxin.

19 It is a double-blind, placebo-controlled
20 study where two-thirds of the patients get treated
21 with Tovaxin and one-third of the patients are
22 treated with placebo.

23 Double-blind means neither the patients
24 who are being given the vaccine or the physicians who
25 are running the study know who is getting the active

1 treatment and who is not.

2 In order to preserve the blind, the
3 person who is doing the vaccine injection is not part
4 of the study team. So, therefore, the doctors don't
5 have a way to know who is getting the vaccine.
6 Because it is an experimental therapy,
7 the study must be monitored by some group who, at
8 least if they needed to, would be able to know who
9 was getting the real treatment and who was getting
10 the placebo. That group is called the Disease Safety
11 and Monitoring Board, and I am the Chairman of that
12 board.

13 So we meet by phone on a periodic basis
14 and review the data from the study to ensure that the
15 study is being safely conducted and that there is no
16 reason to believe that the Tovaxin is causing any
17 harm. If there was harm that was occurring to those
18 patients who were being treated, then we would be --
19 then we would either stop the study or change it.

20 If there is harm to an individual
21 patient because they are doing poorly, the principal
22 investigator at each site can pull any patient out of
23 the study, it really doesn't matter if they're on
24 placebo or on the active drug, but we are to ensure
25 that the entire study is going on in a safe manner.

1 QUESTION: What are the end points in
2 multiple sclerosis trials?

3 DR. CLYDE MARKOWITZ: Whenever -- in the
4 MS clinical trial arena we've looked at markers for
5 disease activity, and at this point, you know,
6 historically in the past, the primary end points we
7 used, at least in these three clinical trials, has
8 been relapse rates.

9 The reason why we do that is because
10 it's very objective measurable information. You can
11 count the number of relapses a patient has during a
12 trial, you can compare the placebo group to the
13 active drug group, and you can make a statement and
14 say your treatment affected the relapse rate by 50
15 percent or something.

16 The problem with doing that metric in
17 clinical trials is that it takes at least a couple of
18 years for you to gather enough information that will
19 allow you to measure it because the average rate of
20 attacks may only be one attack per year and a half or
21 so. So you need something that's a little bit more
22 frequently, you know, going to occur for you to be
23 able to measure it.

24 So MRI has evolved over the last 20
25 years or so as a very useful surrogate marker of

1 disease activity in MS, and in particular we've used
2 what's known as the gadolinium-enhancing lesion where
3 you can actually see areas of active inflammation in
4 the brain, you can quantitate it numerically, you can
5 also look at volume of lesions and you can say let's
6 count the number of lesions in relation to a
7 treatment.

8 And what we've actually seen where we've
9 done serial studies of MRI, even on a monthly basis,
10 is that the disease is way more active radiologically
11 than it is clinically where you may end up seeing
12 many more, maybe five to ten times more frequent
13 gadolinium-enhancing lesions than you do clinical
14 attacks.

15 So all of our Phase II trials these days
16 are using MRI as the marker of disease activity in a
17 relatively short time point, so you can actually do a
18 one-year clinical trial and be able to capture enough
19 data in that one year on MRI that it would take you
20 two years to get on a relapse from that point.

21 QUESTION: What is the potential for a
22 selective T-cell therapy?

23 DR. CLYDE MARKOWITZ: When you think
24 about some of the trials that have been done to date,
25 including compounds like Tysabri or Campath, we know

1 that if you can prevent these T-cells from getting
2 into the brain, you can either do that by blocking
3 entry or by actually depleting them from the
4 periphery, you end up getting a significant benefit
5 in terms of disease activity.

6 You can see it shut down MRI activity.
7 It can shut down clinical relapses to a very large
8 extent. So we know that these T-cells are, so to
9 speak, pathogenic, they are involved with
10 pathogenesis, they are definitely going to be causing
11 tissue damage.

12 So one of the strategies that you want
13 to look at is, well, can you selectively get rid of
14 these myelin-reactive cells and not necessarily give
15 you a complete blockage of all cells, because you
16 will need to have some surveillance of the brain with
17 some T-cells, you just don't want them to be reactive
18 to your myelin.

19 So that's where Tovaxin comes into play,
20 that you really are looking at a specific therapy
21 directed toward myelin reactivity and not preventing
22 all T-cells from getting into the brain.

23 When you think about having a
24 suppressive effect that you want to have to suppress
25 these immune cells, there are a couple different

1 strategies you can take. One would be if you knew of
2 a very specific antigen, you could target that
3 antigen, very selective. You could also have a more
4 global approach where you say, okay, let's just
5 suppress the immune system.

6 And when we've done that, you do get
7 some benefits in terms of the global approach, but
8 you also carry with that the side effect of that
9 global immunosuppression. And one of the major
10 issues are going to be what are the risks about
11 certain kinds of infection. We believe that the
12 immune system also plays a role in tumor
13 surveillance, so there may be issues on that side.
14 So we don't really like the idea of a general
15 immunosuppressant approach just because these other
16 safety issues are at risk.

17 Ideally, you want to have something
18 that's a selective approach that does not have
19 widespread immunologic effects, but is very targeted
20 to a specific area of that disease pathogenesis.

21 So if you knew that you had a selective
22 affect on myelin-reactive cells, which would keep
23 this really isolated to just the myelin, it wouldn't
24 have any effects on your liver, any effects on other
25 parts of the body or anything, you would have an

1 approach that would have a much better safety profile

2 because you would not be having the risks associated

3 with other immunosuppressants.

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