CELL THERAPEUTICS INC
Form 425
November 18, 2003

Filed by Cell Therapeutics, Inc.

Pursuant to Rule 425 under the Securities Act of 1933

Subject Company: Cell Therapeutics, Inc.

Commission File No.: 001-12465

The following is a transcript of a presentation that was given by Cell Therapeutics, Inc. at the Credit Suisse First Boston 2003 Healthcare Conference on November 14, 2003.

Mark Augustine: Thanks for sticking around on a Friday. I m Mark Augustine, Biotechnology Analyst at Credit Suisse First Boston. It s my pleasure to introduce the next speaker, Cell Therapeutics. And we re joined at the podium by Jim Bianco, Chief Executive Officer. And joining him for the breakout session will be Leah Grant, Investor Relations. Thanks so much.

Jim Bianco: Thank you, Mark. Good morning. Before I get started, as is typical with presentations of this variety we will be making some forward-looking statements. And as such we refer you to our SEC filings for more information about the company s programs and its products. As you may also know, we have an open registration statement with the SEC regarding our merger with Novuspharma, and you can find more information on their website about that transaction.

What I m going to walk through today is our pipeline. Specifically as you know we have a marketed product called TRISENOX®. That product is actually gaining some very nice traction in hematological disease. We ll share with you our plans for label expansion in other disease entities and the timing for those significant milestones. The guidance this year of $24 million in net

sales is on target to make that accomplishment, and be a hundred percent compounded annual growth over the last three years for that product.

We also have, what we believe, is one of the most commercially attractive late-stage oncology pipelines, including our XYOTAX product which is now completing its phase three trials. That you ll hear our first phase three in lung cancer was scheduled to be completed at the end of December. And we anticipate that we are going to beat that target by at least a month.

Pixantrone is now is phase three studies in Europe and about to start phase three in aggressive lymphomas. And we ll share with you some very exciting data that is being presented at ASH in so-called CHOP failures.

And then lastly as you ll hear next week, 2106 is our second polyglutamate conjugative product to enter into clinical studies. This is a camptothecin that has done very well in its phase one trial and it should be entering phase two studies in esophageal and small cell lung cancer early next year.

As we noted on our quarterly financial call, we have a very strong balance sheet with over $225 million in the bank. And as you also saw from our recent release, the shareholders approved the merger between CTI and Novuspharma.

This just tries to demonstrate the depth and the breadth of our pipeline. A lot of shots on goal, if you will. Since most of these products are approved versions of existing marketed agents, we feel that the development risks for this program are substantially different than when one goes in with new chemical entities.

Let s talk a little bit about TRISENOX®. The initial label indication, relatively narrow, acute promyelocytic leukemia. The interesting aspect about this product with that disease now with three years of follow-up data we can claim that we cure about 50 percent of the patients who ve had relapsed/refractory APL. We re now moving this into a variety of other indications, including front-line APL. And very nice data being presented at ASH from MD Anderson that you can replace chemotherapy in the front-line treatment and cause molecular emissions in 80 percent of those patients. They re long-term and durable and obviously that has some significant growth implications for this product and that disease. We re also investigating the product in both MDS specifically the high risk population and in myeloma and several solid tumors.

If you look at our performance over the three years since we launched this product, we re putting up as we said we made $24 million this year. This is our net sales guidance, that we ll make this a profitable operating business. That was our goal three years out to have this pay for itself, for a commercial

organization. And going forward, you look at the estimates in 04 as I mentioned on our conference call we typically will do that at the Q4 financial release. But just giving a sense of where the street s numbers are, they can be a low of $27. Obviously we re going to be well over that run rate just coming into the fourth quarter of this year, versus a high of $43 million. So we think that this continues to be a very attractive opportunity for us as our first commercial product and provides us some discretionary cash flow back to the development organization to help fund our other products in our pipeline.

Look at where the majority of those are being generated. This was the first nine months last year versus the same period in 03. And you could see that MDS now makes up the majority of our sales. About 15 percent are so-called on label sales. We did see a little softening in the myeloma usage. This was really reflected by the launch of Velcade. I can tell you that that has now plateaued off and we re starting to see again a healthy up-surgence in the use of TRISENOX® in multiple myeloma.

Our label expansion strategy is pretty straightforward. This drug does not have traditional chemotherapy-like side effects. In fact, it s not additive when given with other agents. And it is clearly synergistic when you put it together with chemotherapy or radiation therapy. So, no overlapping toxicities, but enhances the signal of the underlying agent. And the diseases that we re

targeting for label expansion, you ll see some data at ASH in two large studies, about a hundred patients each. Looking at the so-called high-risk population. We think that the response rate of durable responses of about 30 percent is probably a good hurdle to get in. Along with obviously transfusion independence across multiple blood cell types meaning platelets and white cells. And probably the most important kind of progression in ultimate leukemic transformation appears to have been impacted in those studies. And we look forward to presenting that data at the upcoming hematology meeting.

This is data from an abstract from Jim Berenson s group in myeloma. You can see that even in patients who fail melphalan, thalidomide, and Velcade, if you add low doses of chemotherapy together with arsenic and (arsenic trioxide) TRISENOX®, you get a very high rate of response. These responses are very durable. And in this population of patients that typically are very poor-risk meaning they have renal failure at the time that they are treated they had a hundred percent improvement in their renal functions in the five of the five patients who had significant renal dysfunction.

We re also investigating based upon the Stanford study the utility of radiation in combination with TRISENOX® in glioblastoma. There are studies that are ongoing with Taxotere® in prostate cancer. And lastly, as I mentioned, we ll

highlight this at ASH a study from Dr. Estey and his colleagues being able to replace chemotherapy in the front-line treatment of APL.

Move on to our second product in our hematological pipeline, if you will. This is a product that we will have acquired through our merger with Novuspharma, called Pixantrone. You may be familiar that this class of agents so-called DNA intercalators are a very widely-used kind of cornerstone therapy in the treatment of diseases such as breast cancer, front-line breast cancer, in front-line leukemias, and lymphomas. And the reason for that is that anthracyclines in that setting can cure up to 30 percent of each of those three disease populations. The major limitation for this class of agents is that if you re not cured i.e., the 70 percent of patients who ultimately will relapse with those diseases they cannot be re-exposed to the class of agents because of the high incidence of cardiac toxicity. And so an agent that has less damage to heart muscle tissue potential such as Pixantrone may allow you to use it really in the prevalence market, which is repeat therapy. And equally importantly it may allow you to put it in combination with agents such as herceptin, where those toxicities currently limit its use. And if you re not familiar with that, herceptin by itself has a baseline cardiac toxicity of about five percent. If you add herceptin in front-line breast cancer with an anthracycline which is a cornerstone agent for that disease, it s an unacceptably high rate of cardiac toxicity. Which is why herceptin really got, I don t want to say niched, for a $600 million, $700

million dollar product. But it got forced play in the so-called salvage second-line population, such as paclitaxel in relapsed breast cancer, in combination with herceptin. So we think that this represents a real upside in the Pixantrone opportunity.

If you look at what our colleagues at Novuspharma did in particular, they looked at the two marketed agents Doxorubicin and Mitoxantrone identified the core moiety of the nucleus that was responsible for the binding capacity to DNA at the top isomeric which is the target for these agents. And then teased out the sections that were responsible for other cardiac toxicity. And in pre-clinical studies this agent had no propensity to cause cardiac toxicity at effective doses when given chronically over several months, even in animals that had cardiac toxicity induced with Doxorubicin or Mitoxantrone. It did not significantly increase the toxicity when treated with Pixantrone.

So our first target is pretty obvious, non-Hodgkin s lymphoma. As you may know there are two large populations of that disease, the indolent lymphomas which is predominately owned by single-agent therapy with Rituxan. Very little chemotherapy used. This is a group that typically is not cured. A lot of chronic treatment. But again, they don t tolerate side effects very well and since they re not cured, most clinicians like the concept of a low toxicity regiment. If you look at the aggressive lymphomas which is the larger

segment of the non-Hodgkin s lymphoma market it s very clear that aggressive lymphomas can be cured with aggressive therapy, no pun intended. But because of that they will tolerate toxicities in order to try to get long-term, durable remissions and cures. And that treatment right now is CHOP with Doxorubicin, with two agents being Vincristine, Prednisone, and cyclophosphamide. And that regiment can cure about 30 percent of patients with front-line treatment.

However, the majority of the patients relapse. When they relapse they cannot get CHOP again. They can t get an anthracycline -containing regimen, and so they typically get these multi-agent regimens that don t contain anthracyclines. That s actually (inaudible). And there are no agents that approved once patients fail multi-agent second-line regimens. So our treatment strategy is, you demonstrate the quickest route for registration is this core population here, since there are no approved agents. And then also demonstrate in fact that when you add an anthracycline safely to this group you can increase the response rates. And as you ll see from some data, you can actually improve on the CHOP response rates even after they fail CHOP.

This is just a single-slide summary of about 200 patients worth of data that has been presented in phase one and phase two studies with Pixantrone. Notably very low instance of cardiac events, and that is despite the fact that more than 80 percent of these patients have maxed out on their prior

anthracycline exposure. So by definition 80 percent of these patients, it is contra-indicative meaning you re not permitted you re not advised to give an anthracycline because of the high likelihood of significant cardiac failure. The dose on the side effect is neutropenia, which is not a bad side effect to have since this is easily managed with growth factors or the appropriate dose reductions. And it s about 30 to 40 percent of the patients in clinical studies who will get neutropenia. And in fact that is at that instance that is no different than what you would see with Doxorubicin.

The most exciting aspect of what we had reviewed during our due diligence when we were proposing to put the two companies together and our interest in Pixantrone was the really high rate of complete remissions with this agent. In fact, having worked on Rituxan when I was a transplant director up at the University of Washington, that was the only other agent that we saw that would produce complete remissions as frequently as we have seen with this Pixantrone product in particular.

Here s a trial that they did. They reported us in a peer review publication this summer. 33 patients; they failed third-line, fourth-line, and fifth-line. The average was fourth-line of treatment. So, they failed CHOP, they failed multi-agent therapies, and they failed at least one other single-agent or combination therapy. A third of the patients had an objective response. 20 percent were complete remissions. And the time-to-disease progression for

this group still hasn t been met with a median extending out past 11 months, with a range of 6 to 24 months. So, very high, durable response rates. Clearly the best single-agent response and durability response reported in the literature. And you can do a Med Pub search to confirm that.

They also looked at patients who failed the so-called ESHAP regimen. These are patients who failed CHOP remember, in the second-line are typically multi-agents that don t contain anthracyclines because they re not eligible to get them. But this was third-line therapy. And in that study they reported at the ISEH meetings, 55 percent of the patients had a major tumor response meaning shrinkage of their tumor greater than 50 percent. And a third of those had complete eradication on scans of their disease, of the 33 percent complete remission rate in a third-line setting.

And then finally the data that I ll show you a little bit more granularity on in a second, is being reported at ASH. This is off their abstract. These are patients who failed CHOP and then they came in and they got Pixantrone as the secondary exposure to an anthracycline. That is highly unusual since most of those CHOP failures were ineligible to get an anthracycline. And 80 percent of those patients had major tumor shrinkage, and most importantly more than half of them had complete disappearance of the tumor.

53 percent complete remission, as you saw from the ECOG or CHOP data that was recently reported in the ASH abstract, is typically what you would expect to see in the front-line combination therapy treatment of this disease. These are all multi-center studies conducted both in the U.S. and Europe. This was the phase one component. This is kind of a preview of what will be presented at ASH. You can see they did a dose escalation study of Pixantrone in the CHOP regimen, where they replaced the Doxorubicin with Pixantrone. And in a dose-dependent fashion they had a nice increase in response rates, such that at the 150 milligrams per meter square dose every three weeks they had all the patients responding, with five of them having complete remissions and two PRs. Ten out of 19 patients had a complete remission in that phase one component. The phase two component is now enrolling 75 patients and we should see an update on that at ASH.

The clinical trial effort that we intend on continuing and expanding really follows that whole market development line of strategy. We essentially want to own the aggressive lymphoma space very much the way that Rituxan owns the indolent lymphoma space. We think we ve accomplished that with this just an outline of some of the studies that we are pursuing. There are trials looking at it and you ll see this data at ASH as well, in combination with Fludarobine and Rituxan in the indolent lymphoma setting. I think that the tolerance for neutropenia in that population is obviously a lot lower than it is the aggressive lymphoma trials.

And then lastly if we re successful in demonstrating that we can combine anthracycline with herceptin in primary metastatic breast cancer then that obviously has some very significant commercial implications. Just if you look at the break-out of remember, this is a multi-sourced market. This doesn t reflect the units, which is very robust in terms of number of patients and number of unit sales. But again, price erosion on a generic side. It s still a healthy $197 million dollars for the lymphoma and the leukemia applications, predominantly for Doxorubicin. Actually, Mitoxantrone shares very little in leukemia. It s only about $11 million a year. It s mostly a prostate and MS application for that agent. And Epirubicin is the predominant agent used in breast cancer. And so we think that the opportunity here is a significant one, certainly more than the $150 million that we initially projected for peak sales for Pixantrone.

I m going to conclude my discussion this morning updating you on our polyglutamate paclitaxel Cleary this has the ability to transform the company into a significantly profitable entity given the size of the market that the taxanes currently enjoy in the U.S. and Europe. And this is a technology that was developed in MD Anderson Cancer Center. And if you ve been watching all the Genentech collateral material around Avastin this is actually very helpful for us from the clinical education perspective. Because the technology tries to exploit what has been well-known, the fact that the blood

vessels that tumors make are distinctly different than the blood vessels that are normal in your body that your normal tissues have. In particular they re porous. They have large openings in them, and pores. And that allows them to be porous the big molecules, so-called macro-molecules like polymers.

And so one way to potentially target a tumor tissue with chemotherapy is to optimally size a polymer so that as it circulates in the blood stream it gets preferentially trapped in the tumor tissue at a rate that s higher than it does in normal tissue. So now that if you bind the chemotherapy physically bind it to the polymer as the carrier molecule you have two potential benefits. One, you should get more of the active drug going to the target tissue than you do to normal tissue. And theoretically that should give you better efficacy. But because now the chemotherapy is no longer circulating free in the bloodstream it s bound to a polymer in an inactive form, if you will then the acute side effects of Taxol® should be dramatically reduced. Because if you look at the peak level that you get after you give a standard dose of Taxol®, the free amount of paclitaxel coming off the XYOTAX polymer at the same dose and same time is 200 times lower in the blood stream of patients. And what that translates into should be less of the acute side effects, like the allergic reactions, the neutropenia, and the hair loss.

You also have another benefit in that this polymer in yellow carrying the chemotherapy gets into the tumor cell through a well-described mechanism called transcytosis. And that means that instead of it leaking in like most formulations do, you now have a mechanism for the polymer to be uptaken by the tumor cell, bypassing a primary point of resistance. And then once inside the tumor cell or other metabolically active tissues, that polymer gets digested by those enzymes that are abundant in these cells, releasing the chemotherapy inside the tumor cell. So you have a site-selective release of the chemotherapy, which again should increase your specificity for the target over the normal tissues.

And in fact, while this is our targeted product profile in now over 350 patients in our phase one and two studies, this is pretty conclusive of the safety profile of this drug. It s a ten-minute infusion. It, for the most part, does not require pre-medications. I think out of 350 patients we ve had two so-called grade two hypersensitivity reactions even though they weren t pre-medicated. And they required pre-mediation after that, but clearly more than the majority essentially all of those patients tolerate the ten-minute infusion very well. No special fusion kits. We hear this over and over again. It doesn t feel or look like chemotherapy. These patients don t have hair loss. Neutropenia is markedly reduced compared to equivalent doses of Taxol® or Taxotere®. Neuropathy occurs less frequently although we certainly do see neuropathy. And that has to do with the total exposure of the drug over time

as opposed to the acute exposure. And because of this side effect profile this agent appears to be much better tolerated and get more doses in. And that should translate into superior efficacy.

And so our target development program has targeted two of the most frequented populations or diseases of use for taxanes. In specific paclitaxel, as you may know, and as you see from this slide, paclitaxel and carboplatinum is really the standard front-treatment, whether it s early stage three disease with radiation or whether it s advanced stage four disease. Taxol® is king. It isn t Taxotere®. In fact, lung cancer and ovarian cancer make up $498 million of the U.S. sales for Taxol® paclitaxel as opposed to breast cancer and prostate cancer, which are the primary uses for Taxotere®. And so we focused on where the market clearly agrees that paclitaxel is the better agent of choice. And then if you fail front-line therapy Taxotere® is the only approved agent in second-line, although it s infrequently used because of its side effects. Most people tend to focus on gemcitabine or other less toxic agents.

You get a sense of how do patients do if they have advanced lung cancer and they re so-called poor performance statuses are a particularly interesting group for us to target because we believe that if the agent is better tolerated with lower side effects then more chronic treatment of this population may actually extend their survival.

And if you look, their survival is quite poor. These are PS2 front-line therapy and these are from randomized multi-center studies that were recently reported. Gemcitabine is considered to be inactive in this disease. Many times progression is six weeks, and half the patients are dead at 60 days or two months. Paclitaxel is 20 percent better. Actually, it does show a modest improvement in survival. It actually happens to be significantly different at 2.4 months. And then obviously if they could tolerate the double combination of Taxol® and carbo which is the standard you can improve that survival of 4.5 months. But for PS2s this is dramatically a poor outcome. Pac and carbo and front-line therapy for good-risk patients that should be about 10 months for median survival.

So we thought this was a good population to target. We did a phase two study of 30 patients. 28 patients were evaluable. They were very elderly, advanced age 76. About a third of them were also PS2s. So this was a particularly high-risk group of patients. And the first thing that we and our thought leaders noticed was that it was extremely well-tolerated. Only one patient had grade four neutropenia. That was a very low incidence. And importantly half these patients got four or more cycles of therapy. Right there is a signal that if you re tolerating three months of therapy your disease obviously isn t progressing, because you d come off study when your disease starts to progress. And in fact a third we see six or more cycles of therapy.

And that translated into median survivals that were unique for this population. If you even call out the PS2s it was 5.4 months 22 weeks. And if you looked at the better-risk so called PS0s and 1s, 8.8 months is right up there with the double therapy that s reported for both 0s and 1s in the literature.

And as a result of that study as well as about 150 other patients in various phase ones and twos studies in lung cancer applications, we initiated a series of phase three trials called the STELLAR 2, 3, and 4. STELLAR 3 is going to complete as I mentioned probably this month instead of December. 370 patients appear to show an improvement in survival when you give Taxol® in combination with carboplatin, the standard front-line therapy. Versus XYOTAX in combination with carbo. I think the fact the rapidity of enrollment this is mostly a U.S. and Western European study really underscores the experience that we have been seeing from the ease of use of this agent in clinical studies.

STELLAR 4 is single-agent gem versus XYOTAX. I m going to talk about the dose reduction that we recently did in this trial, at least the rationale for it. That study is scheduled to complete its enrollment at the end of the first quarter next year. And then STELLAR 2 the very large, randomized trial versus Taxotere® should complete its enrollment at the end of the second

quarter next year. All of this allowing us to be in position to file our NDA for the first indication on either STELLAR 3 or STELLAR 4 by the fourth quarter of next year. Those two indications are fast-tracked status from the FDA and so we will actually begin our rolling submission sometime in the second quarter of next year.

I mentioned the dose reduction and the justification. If you look at XYOTAX of taxane to taxane, in terms of grade four neutropenia, clearly at 175 the standard marketed dose of Taxol® we have a very low instance of grade four neutropenia compared to Taxol. This is in about 165 patients. So, good sample size for confidence well, that s real. If you go up to 210 we get 11 percent and at 235 it s 27 percent. It s clearly below Taxol® level and below what you see for Taxotere®. This was the maximum tolerated dose in our phase one studies in lung cancer. That was the dose that the principal investigators argued should be put into the study, versus gemcitabine, to maximize the so-called efficacy of the product. Even though we had very good efficacy reported at 175 the data I just showed you.

Well if you look at gemcitabine, as I said gemcitabine is an inactive agent in this disease. It has a very low instance of neutropenia but unfortunately it has no effect on median survival no matter what you do with the gemcitabine dose. And that was recently reported. So we felt that lowering it to 175 would allow us not to see those early neutropenic related events that we saw

in about five or six patients, compared to the comparator arm. And increase our chances that this drug actually will prove superior in effecting the outcome of patients with PS2 versus the gemcitabine arm.

I m going to close by mentioning a little bit about ovarian cancer. A different disease compared to lung cancer. As you can see from here about a third of them live five years or more. So, more of a chronic ailment than it is an acute ailment. Current standard of care for this disease, Taxol® and carbo. Does very well, very high response rate. The complete remission is about a third to almost half. Unfortunately they relapse at about 13 months. If you give maintenance therapy to these patients with Taxol®, you have a highly significant improvement in progression-free survival. Unfortunately Taxol® is not approved for that so it has not been considered to be adopted at this standard. And the reason for that was, monthly Taxol® for a year 70 percent of those patients had dose reductions because of neuropathy, neutropenia, and hair loss.

So as you ll see the GOG is now proposing we re initially discussing doing a non-inferiority study comparing Taxol® and carbo to XYOTAX and carbo front-line. And what the GOG has discussed with the FDA and when we get final signoff from the FDA is doing a XYOTAX versus no-maintenance/maintenance study. To try to reduce these toxicities and to show that you can replicate that highly significant improvement in progression-free

survival. Obviously if the FDA signs off on that we think that that would be a very big shoe-in as a win for us with this product and ovarian.

This is some data just demonstrating that in phase one, in about 44 patients. Good response rates in ovarian. Good duration of response is 30 weeks with the phase one population. So highly durable responses and chemo-resistant disease. Even as a single-agent, 175 the standard dose again, pretty robust. 100 patients well tolerated, low instance of neutropenia. And again, no hair loss, and only one of those patients had a hypersensitivity reaction.

This was the initially proposed GYN oncology group trial. We ll update you in the near future with the current plan from the FDA, assuming that the FDA signs off on that direction. But obviously very exciting for us if the GOG has actually come up with a very novel approach to getting that drug approved in ovarian cancer.

These are our timelines, as previously stated. Either the STELLAR 3 or STELLAR 4 will be the first basis for an NDA. That will happen in the fourth quarter of next year in terms of the completion of the filing. And then the first half of 05 for the approval under the fast-track regulations.

Next three to six months, we re just giving you a short-term outlook of what obviously are some significant regulatory and clinical milestones for us. Out

from the GOG/FDA meeting and then their initiation of the phase three trial. That will be the first time that a cooperative group has negotiated a study with the FDA and will conduct an investigational trial in concert with CTEF at the NCI, the academic realm and the regulatory realm for a drug company s investigational drug.

As I already kind of alluded to, we will announce the completion of our phase three trial enrollment with XYOTAX well ahead of schedule. We know that Pixantrone will qualify for the so-called accelerated review track. When we initiate that study in the first quarter we will make that announcement.

We also have a strong basis to believe that we can now extend our TRISENOX® franchise beyond the regulatory exclusivity which ends in 2007 to a patent exclusivity that would end in 2018. Really allowing us to view that product very much like a thalidomide type of revenue potential for us. And then certainly ASH in the near term will be a very highly visible program with data on Pixantrone and TRISENOX® in well over 30 presentations between the two products combined.

And then lastly, we should hear from the CONSOB in December about our listing, which was the final condition for the Novuspharma merger, which we essentially are working on as an integrated company today. Thank you for

your time and attention. Mark, thank you for having us here at your conference.

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CAUTIONARY STATEMENT REGARDING FORWARD LOOKING STATEMENTS

This transcript contains forward-looking statements within the meaning of the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. These statements are based on management s current expectations and beliefs and are subject to a number of factors and uncertainties that could cause actual results to differ materially from those described in the forward-looking statements. The forward-looking statements contained in this transcript include statements about future financial and operating results, the proposed CTI/Novuspharma merger, and risks and uncertainties that could affect CTI s product and products under development. These statements are not guarantees of future performance, involve certain risks, uncertainties and assumptions that are difficult to predict, and are based upon assumptions as to future events that may not prove accurate. Therefore, actual outcomes and results may differ materially from what is expressed herein. For example, if either of the companies fail to satisfy conditions to closing, the transaction will not be consummated. In any forward-looking statement in which CTI expresses an expectation or belief as to future results, such expectation or belief is expressed in good faith and believed to have a reasonable basis, but there can be no assurance that the statement or expectation or belief will result or be achieved or accomplished. The following factors, among others, could cause actual results to differ materially from those described in the forward-looking statements: risks associated with preclinical, clinical and sales and marketing developments in the biopharmaceutical industry in general and in particular including, without limitation, the potential failure to meet TRISENOX® revenue goals, the potential failure of XYOTAX to prove safe and effective for treatment of non-small cell lung and ovarian cancers, the potential failure of TRISENOX® to continue to be safe and effective for cancer patients, determinations by regulatory, patent and administrative governmental authorities, competitive factors, technological developments, costs of developing, producing and selling TRISENOX® and CTI s products under development in addition to the risk that the CTI and Novuspharma businesses will not be integrated successfully; costs related to the proposed merger; and other economic, business, competitive, and/or regulatory factors affecting CTI s and Novuspharma s businesses generally, including those set forth in CTI s filings with the SEC, including its Annual Report on Form 10-K for its most recent fiscal year and its most recent Quarterly Report on Form 10-Q, especially in the Factors Affecting Our Operating Results and Management s Discussion and Analysis of Financial Condition and Results of Operations sections, its Current Reports on Form 8-K and its filings on Forms S-3 and S-4. CTI is under no obligation to (and expressly disclaims any such obligation to) update or alter its forward-looking statements whether as a result of new information, future events, or otherwise.