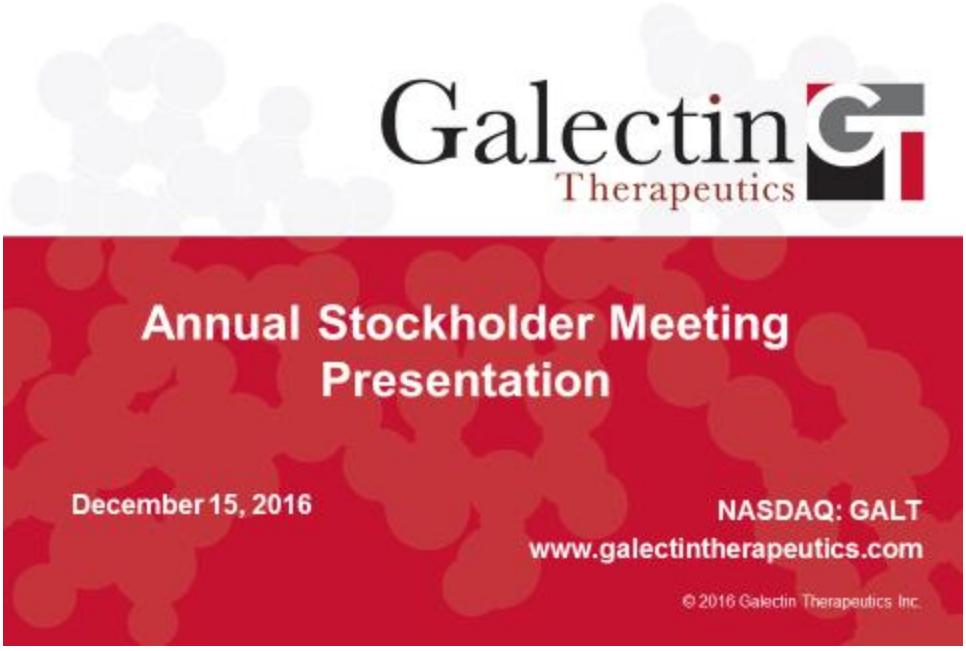

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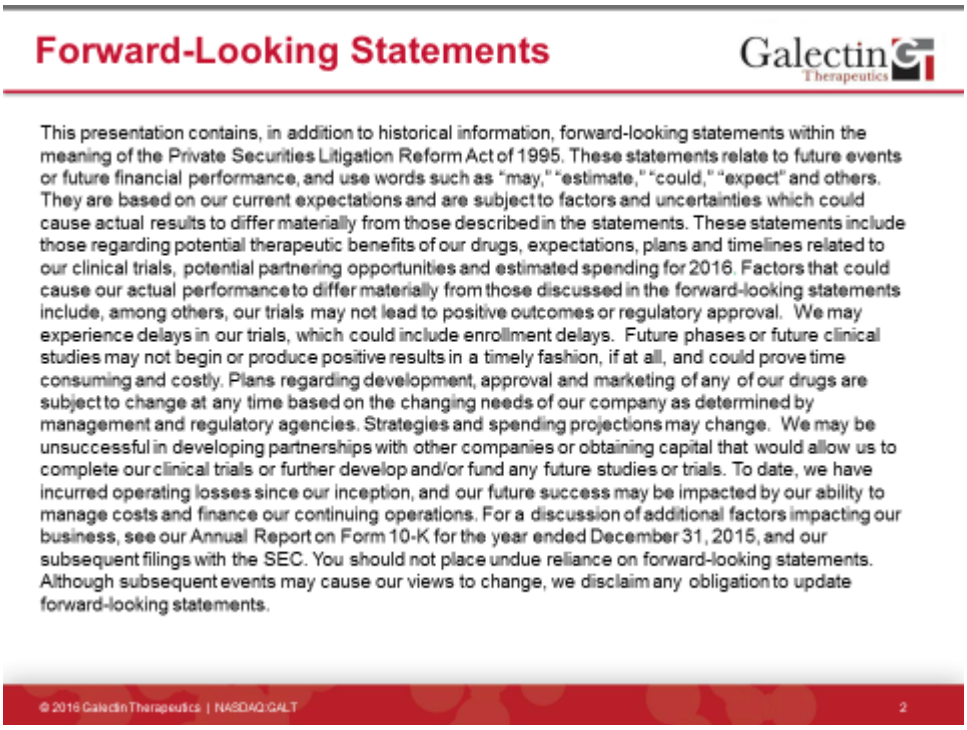
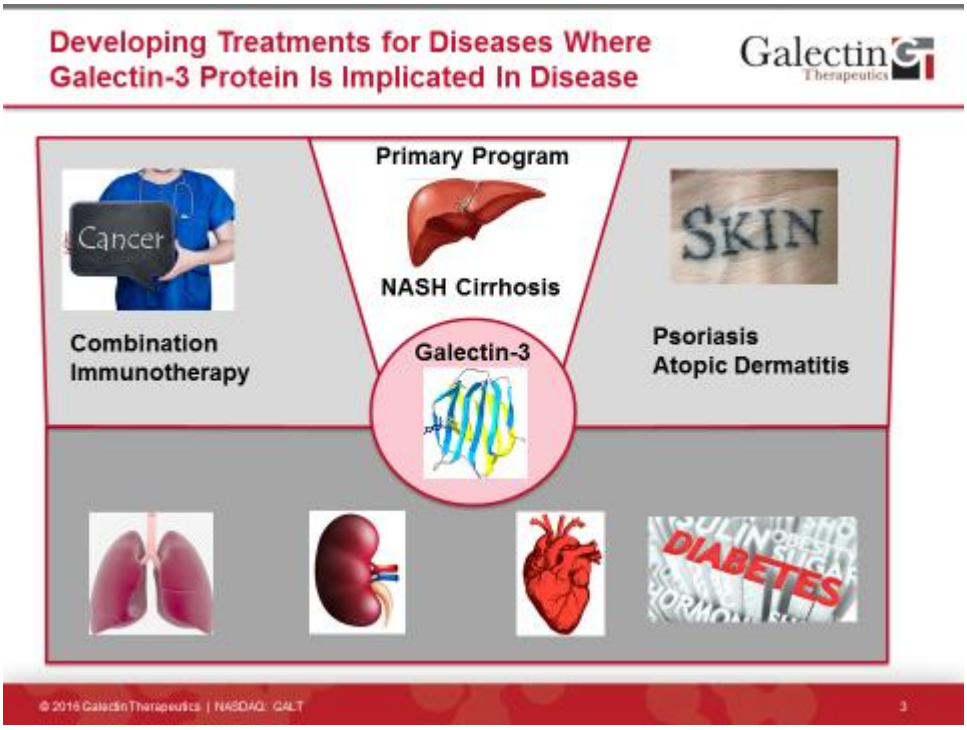
My name is Dr. Peter Traber the CEO, President and Chief Medical Officer at Galectin Therapeutics. Welcome to the Annual Stockholder Meeting Presentation that is following the official stockholder meeting. I want to welcome everybody who is in the room and on the phone.



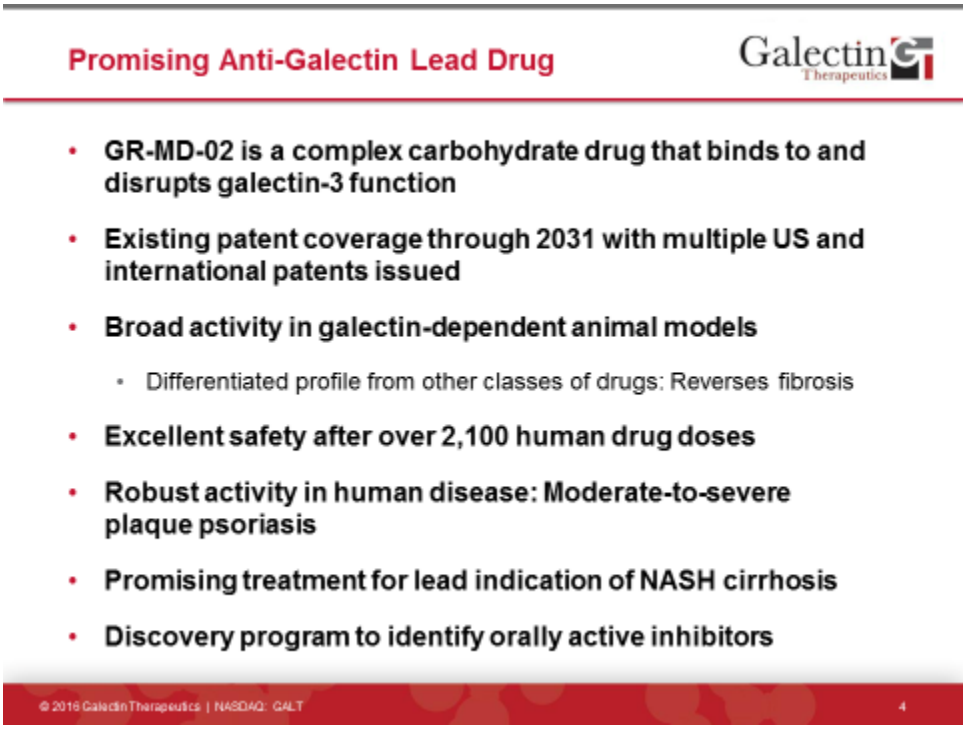
Statements that we will make in the following presentation and in the question and answer session will be forward-looking statements, statements that look forward in time or that are expressing beliefs or expectations regarding the future. They are forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. These forward-looking statements are subject to a number of risks and uncertainties that may cause actual results to differ materially from current expectations. For a discussion of these risks, please see the risk factor section of Galectin Therapeutics' Annual Report on Form 10K for fiscal year ending December 31, 2015, and our subsequent filings with the SEC.

I would ask that those of you who are on the line to mute your phones or the device that you are listening on.



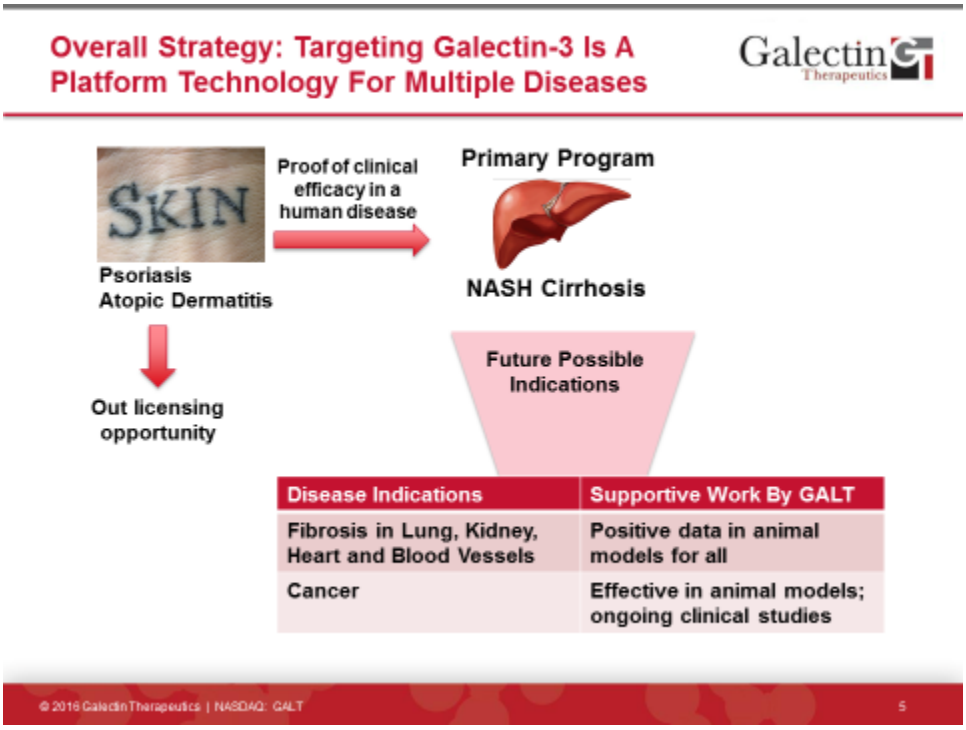
As a company, Galectin Therapeutics is developing treatments for diseases in which the galectin-3 protein is implicated in the disease. This is a very important point. The galectin-3 protein is made by a number of different cells in the body but predominately by immune type cells, cells that protect the body and can cause damage when they are over-stimulated. An increase in galectin-3 in multiple diseases is the cause for pathological damage in a number of these diseases. Our primary program is in liver disease, particularly NASH or fatty liver disease, with chronic damage resulting in fibrosis and finally advanced scarring called cirrhosis. That is our primary program. However, we have ancillary programs that are in clinical trials that include the skin diseases psoriasis and atopic dermatitis, both of which are immune mediated type diseases, and cancer combination immunotherapy, and I will tell you about how galectin-3 is involved in the immune system and cancer. Also, I want to mention that there are multiple other diseases in the lung, kidney, heart and even diabetes that are in part dependent on the galectin-3 protein.

The Company is focused on galectin-3 and fatty liver disease and cirrhosis, but there is a broad spectrum of diseases that galectin-3 is involved in.

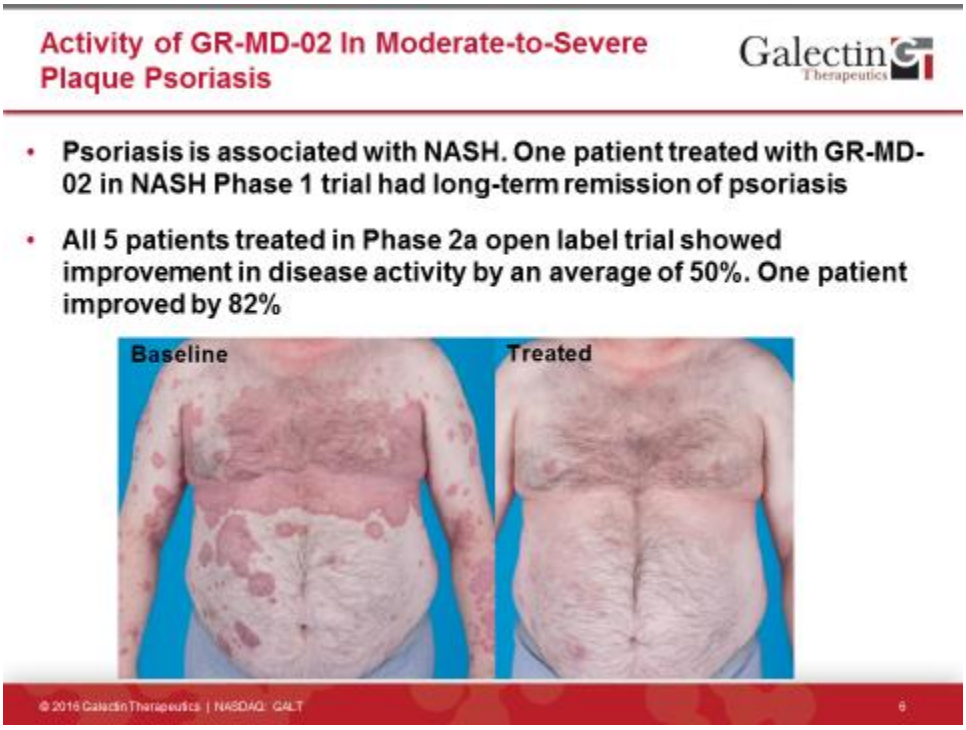


The reason why this is important for us is that we have a very promising anti-Galectin lead drug. Our drug, GR-MD-02, is a complex carbohydrate, or complex sugar, that binds to and disrupts the function of galectin-3. We have strong existing patent coverage through 2031 and this drug has shown broad activity in galectin-dependent animal models of disease. In fact, we have a differentiated profile over other classes of drugs that are focused on the same things we are. For instance, we have reversed fibrosis in animal studies, and I will talk about that more. We have excellent safety after over 2,100 human doses of our drug. We have robust activity in a human disease -- which is moderate to severe plaque psoriasis -- and we have promising treatment for our lead indication of NASH cirrhosis.

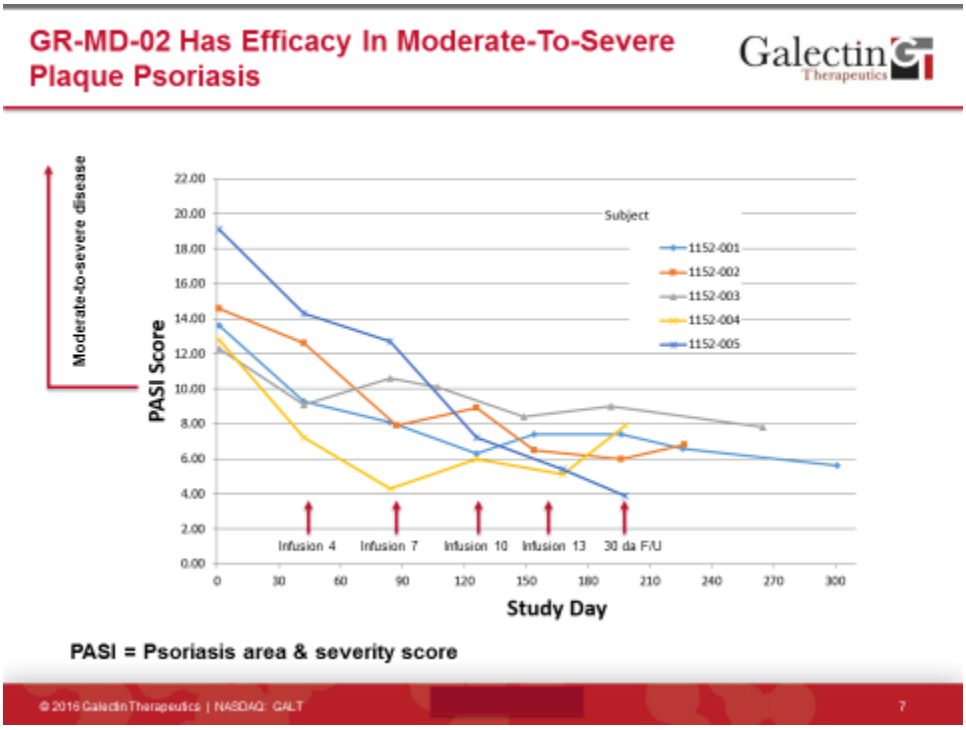
Finally, I want to mention we do have discovery programs to identify orally active inhibitors which could be very important as follow-on drugs. Therefore our overall strategy is targeting galectin-3 that may be a platform for multiple diseases.



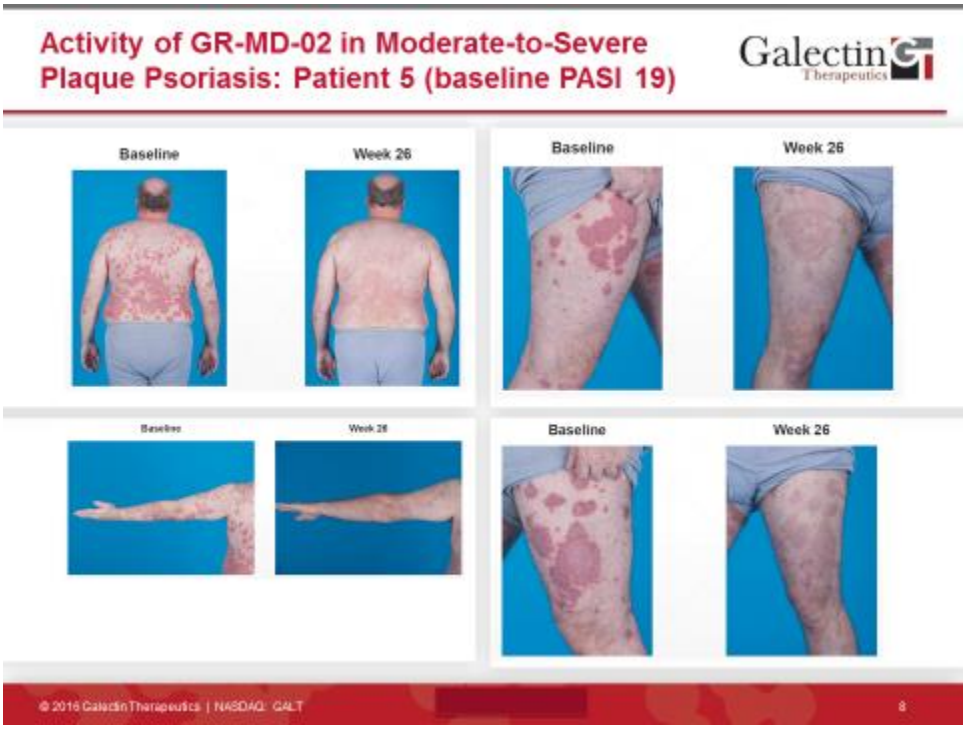
Our primary focus is NASH Cirrhosis which I'm going to talk about extensively. But I want to point out that psoriasis and atopic dermatitis, two important skin diseases are diseases that we have also focused on. I am going to tell you about the data, and this may be used as a proof of clinical efficacy in human disease in support of our primary program. Furthermore, this presents an out-licensing opportunity which I'll also describe. Finally, there are multiple future possible indications. Fibrosis of the lung, kidney, heart and blood vessels are scarring disease, and we have positive data with GR-MD-02 in multiple animal models for all of these. Second, in cancer, we have shown that combination of our drug with cancer immunotherapy is effective in animal models, and we have ongoing clinical trials in that, and I will talk to you more about it. The primary point of this slide is to orient the stockholders to the potential broad importance of galectin inhibition and the possibilities that exist for therapeutic targets.



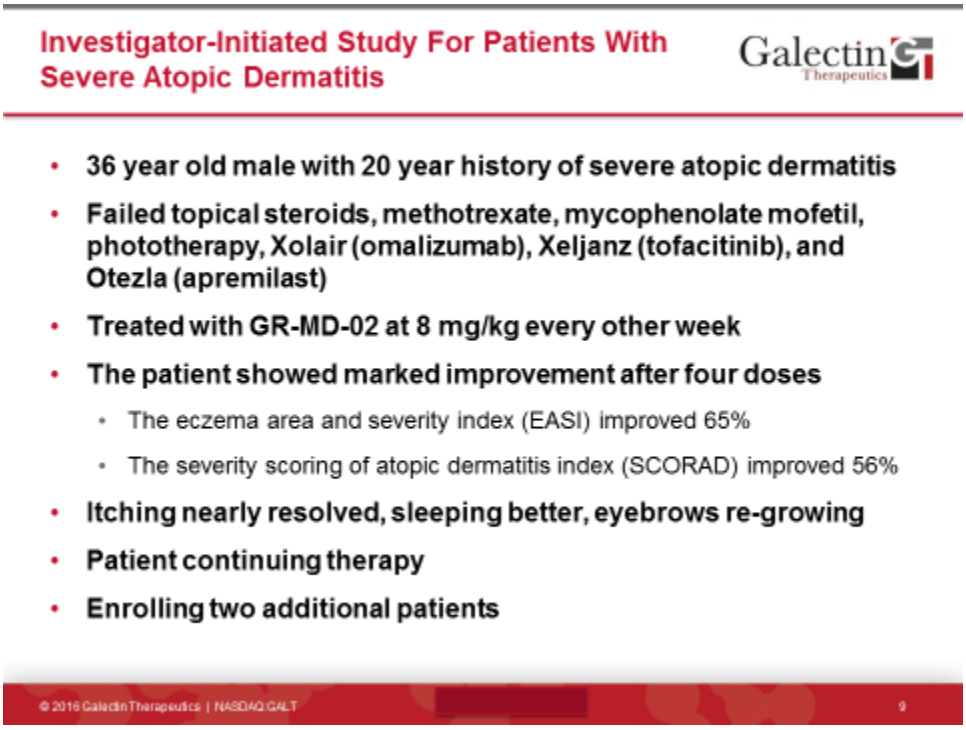
I have changed around the presentation a little bit from what you may have heard before because I'm going to talk about skin disease first and then talk about our primary program, NASH Cirrhosis, because I think proof of concept in a human disease is a very important point for our clinical programs. We have shown that there is activity of GR-MD-02 in moderate to severe plaque psoriasis. This came about by serendipity. One of the individuals in our Phase 1 one clinical trial in NASH happened to have psoriasis, and that patient got better on GR-MD-02. Therefore, we initiated an open label Phase 2a study in patients with moderate to severe psoriasis, and we planned on doing 10 patients at a single site. We ended up doing five patients, primarily because that site could only readily identify five patients and we would have had to expand to other sites, expending additional funds, in order to recruit 10 patients into the trial. However, it was very encouraging that all five patients in this trial showed improvement in disease activity by an average of 50%, and, in fact, one patient improved by 82% by the end of the trial. I show a picture of this individual on this slide, and you can see the extensive psoriatic disease that he had had for many years.



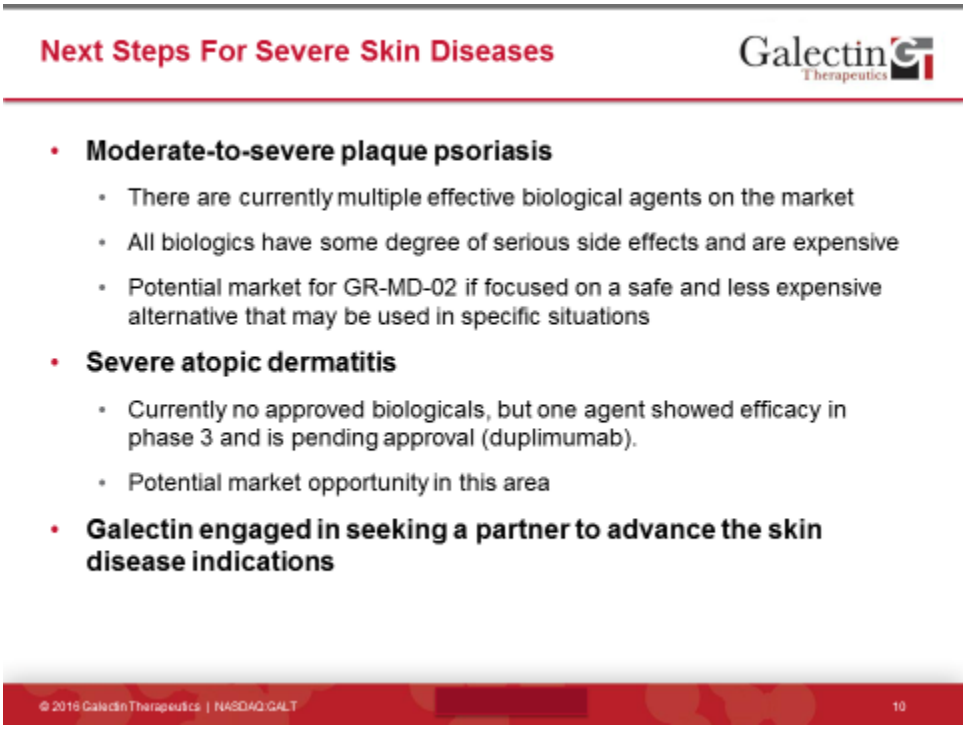
This graph shows all five patients that we treated with moderate to severe plaque psoriasis. You can see from the PASI score which is the psoriasis area & severity score along the Y axis, all of the patients had over ten on this scoring system, which is moderate to severe psoriatic disease. In fact, they all had over 12. And you can see the marked improvement after 13 infusions and a 30-day follow-up. The one patient I showed you before, who had the highest severity of psoriasis, decreased by over 80% by 30 days following his last infusion. So, a very clear and dramatic response. Experts in psoriasis and individuals who are dermatology experts will tell you that spontaneous placebo-dependent responses in psoriasis are not seen. This is clearly an important result.



Just to show you more pictures from that one person who improved over 80% showing his back, his right leg, his left leg, the back of his arm. These are really important and clinically relevant improvements in these patients with moderate to severe plaque psoriasis.



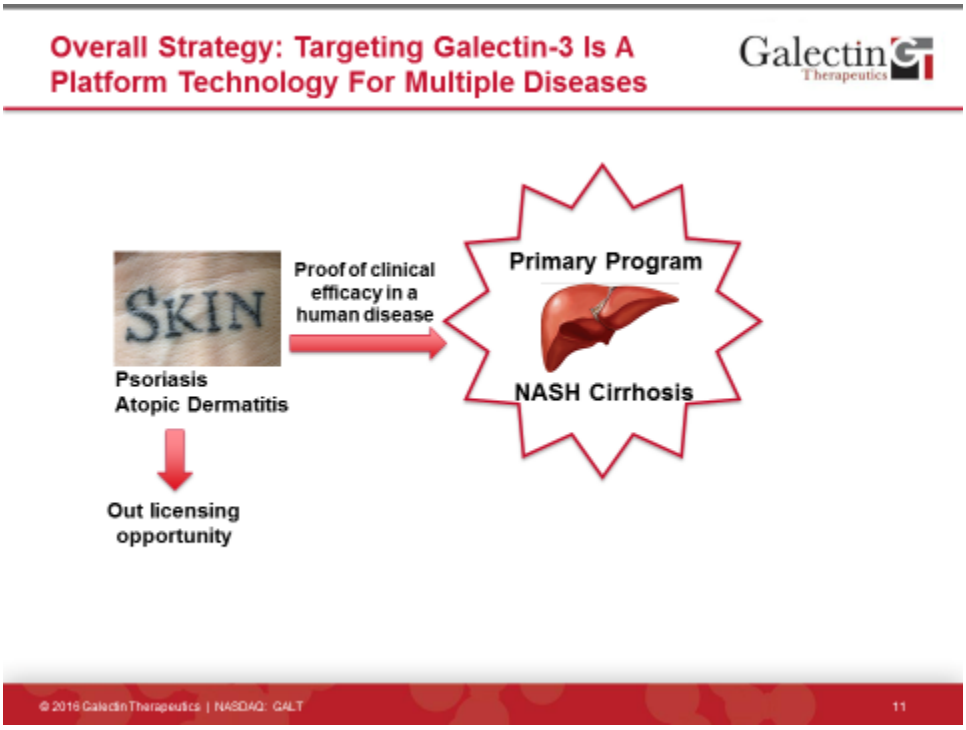
The same investigator who was doing this trial with plaque psoriasis also treated a person with severe atopic dermatitis in an investigator-initiated study. This was a 36-year-old man with a 20-year history of severe atopic dermatitis and had been on seven different types of drugs and therapies for treating this disorder, and he had failed all of them. He was treated with GR-MD-02 at 8 mg every other week, and the patient showed marked improvement after four doses. His eczema area and severity index, called EASI, improved 65% and the severity scoring of atopic dermatitis index, called SCORAD, improved 56%. His itching nearly resolved, he is sleeping better and his eyebrows are re-growing from reduced scratching. This is after a 20-year history of suffering with this disease. A very impressive result to the investigator, Dr. Simon Ritchie. That patient is continuing therapy, and, last week, two additional patients were enrolled under this investigator-initiated trial. Sometime in early 2017 we will have continuing results on three patients with severe atopic dermatitis.



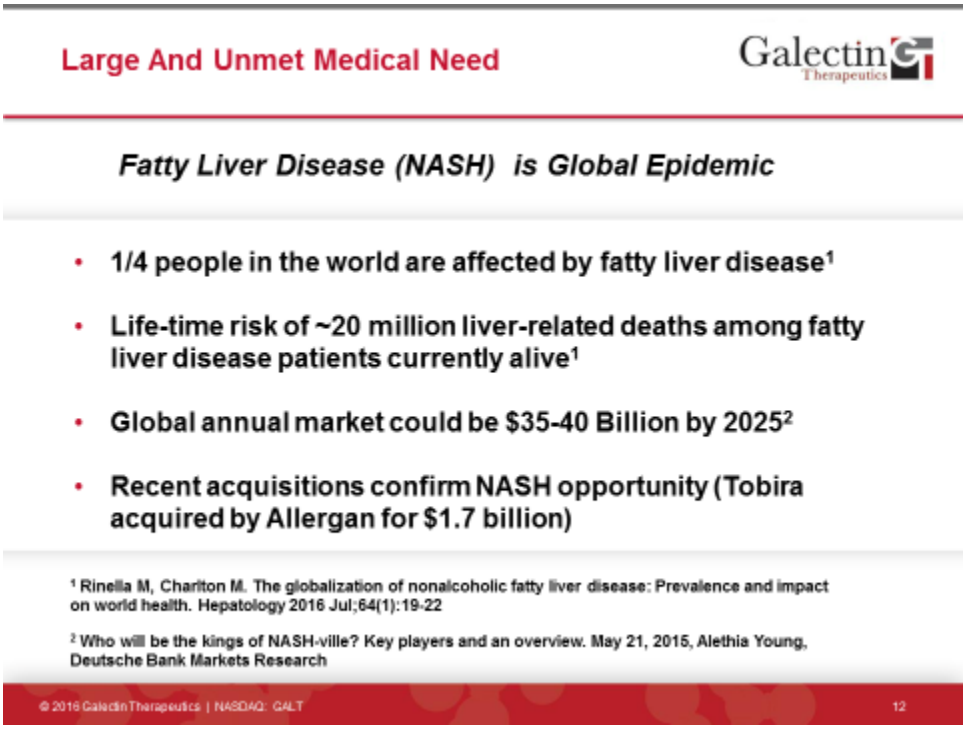
What are the next steps with skin diseases? It is an important issue why are we talking about skin diseases again now and how it impacts the rest of our programs. For moderate to severe plaque psoriasis, there are multiple effective biological agents on the market. All the biologics do have some degree of serious side effects and are expensive. Nevertheless, there are lots of effective drugs, resulting in a significantly crowded market. There is potential for GR-MD-02 if it is focused on a safe and less expensive alternative that could be used in specific situations. But that crowded market does make it challenging for new psoriatic drugs delivered by a parenteral route.

However, for severe atopic dermatitis, there are no currently approved biologicals for this indication. One agent has shown efficacy in phase 3 and is pending approval, *duplimumab*. But, that is one agent that is still not approved in a potentially very large market. This provides a potential market opportunity for a drug like GR-MD-02 which may be an important approach.

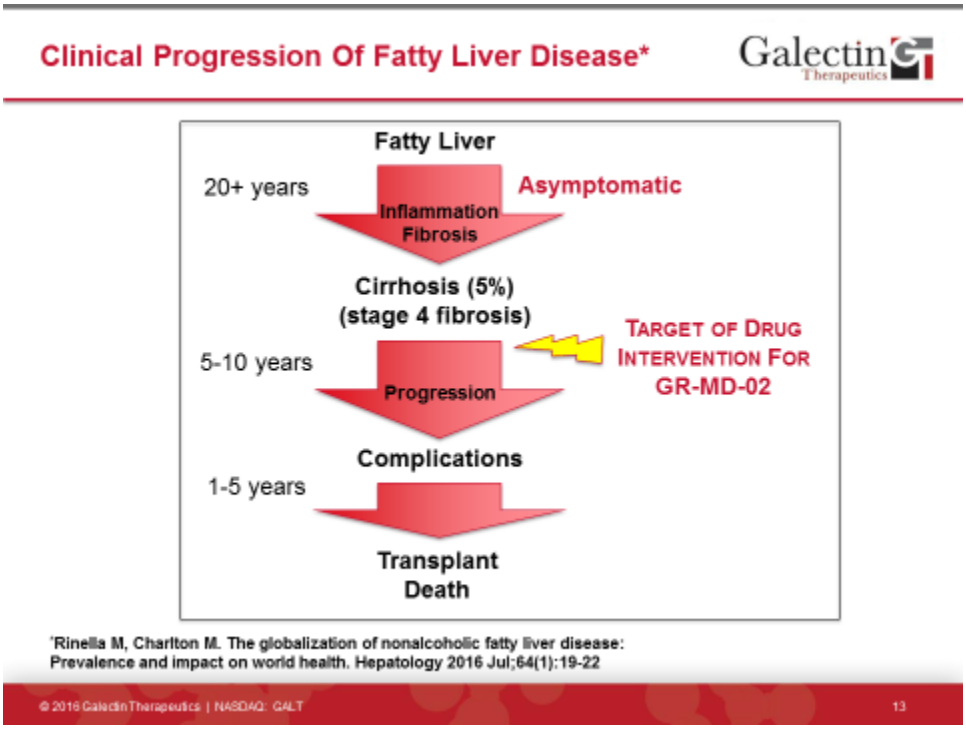
We are engaged, as we reported in a press release, in seeking a partner to advance the skin disease indications. We will need a partner to both fund and drive forward skin disease indications, and the important aspect about where we stand now versus several months ago, is the effect in atopic dermatitis increases the potential interest in this agent in skin diseases. We are excited about that. We'll keep the shareholders up to date on that as that investigator-initiated trial continues.



Let me come back to our lead indication and primary program, which is NASH Cirrhosis. I presented to you the skin disease program. It is important to note that we have shown an important effect in a human disease, which does not directly tell us that our NASH program is going to work but shows there is an immune-mediated disease where it works, and there is a lot of immune mediation involved in NASH as well, so there is a possible interaction. This is why I presented you the skin disease data and potential for out licensing, but now I want to talk about our primary program, NASH Cirrhosis.

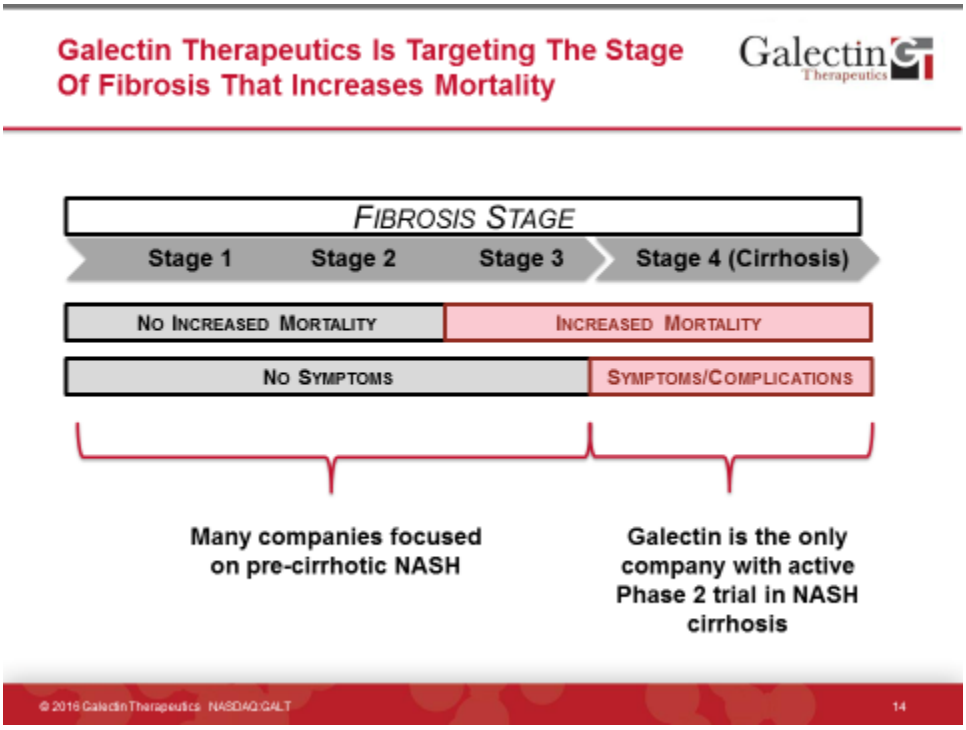


Fatty liver disease is a global epidemic and a very large unmet medical need. In fact, one in four people in the world have fatty liver disease, a staggering number. The life-time risk is about 20 million of those patients who currently have fatty liver disease will die liver-related deaths. Of those people in the world that have fatty liver disease right now, about 20 million of them will die as a result of their fatty liver disease. It is estimated by Deutsche Bank that the global annual market for fatty liver disease could be as high as $40 billion by 2025. Recent acquisitions confirm that the NASH opportunity is large. Tobira was recently acquired by Allergan for $1.7 billion based on preliminary Phase 2 data.



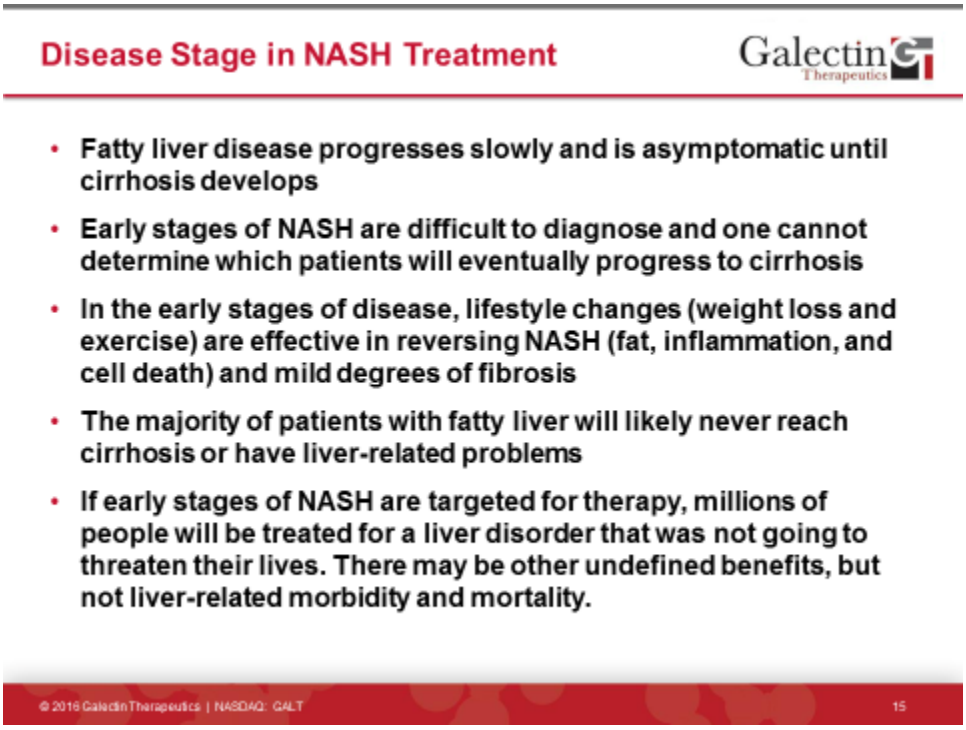
In order to recognize where Galectin Therapeutics is in this field, it is important to understand the clinical progression of the disease. Fatty liver disease occurs when fat develops in the liver and you get inflammation and fibrosis or scarring and that goes on for a long time. It is a chronic disease, up to 20 years of which is completely asymptomatic. At some point, some of these patients, 5% of the total, develop cirrhosis, or stage 4 fibrosis. Cirrhosis progresses over five to ten years, and patients start having complications. Once they develop complications, within one to five years they either need to receive a liver transplant, or they succumb to their liver disease.

While it's only 5% of people who develop cirrhosis, remember the denominator is huge, one in four people in the world. It is really critical in a chronic disease that progresses over this period of time to decide where you are best suited to intervene and where it is most important to intervene. The targeted intervention for our drug, GR-MD-02, is in well-compensated cirrhosis. These patients have cirrhosis, but they have not experienced complications of cirrhosis and are still at low risk for imminent transplant or death. They still have the possibility to have their cirrhosis reversed and to maintain their health.



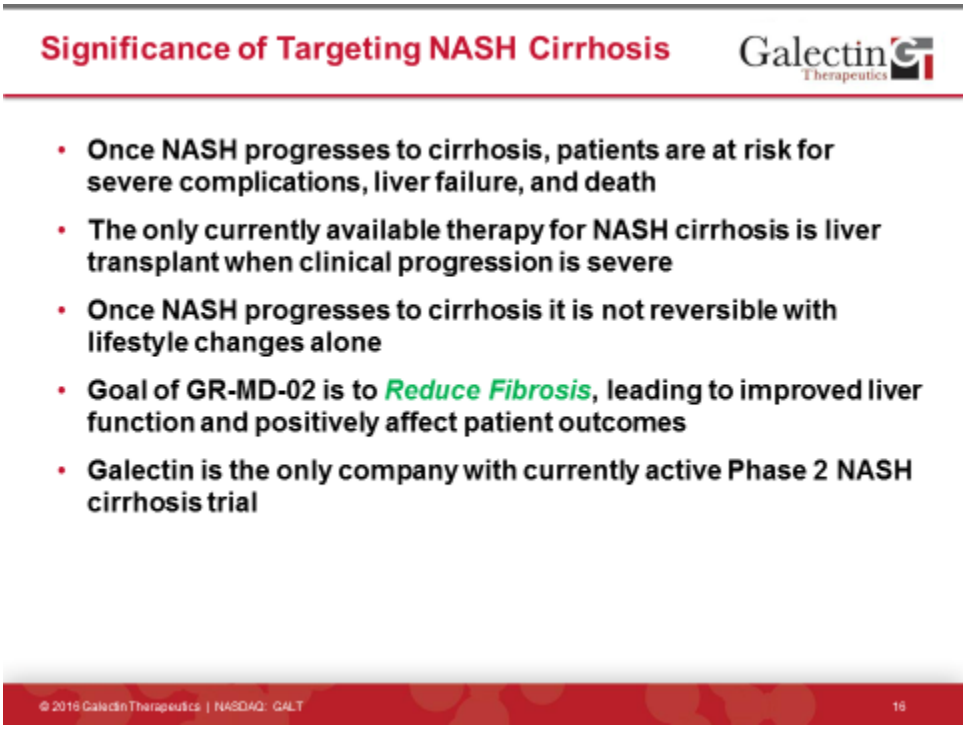
Fibrosis staging is characterized in four stages in NASH. Stages one through three, which I won't go into detail with, but then Stage 4 is cirrhosis, and this is a really important critical step going from fibrosis to cirrhosis because that is when patients have problems. It has been well known that for early stage fibrotic disease in NASH, there is no increased mortality. If you follow those patients over 33 years, there is no increased mortality over the reference group. However, if you look at Stage 3 or Stage 4, there is a marked increase in liver-related mortality. The severity of fibrosis is a critical factor. Furthermore, there are no symptoms really at all of Stages 1 through 3. And then, of course, at Stage 4 there are symptoms and complications.

What is important about this from a drug company standpoint? Well, many companies are focused on pre-cirrhotic NASH, and most of you will know companies that are focused on pre-cirrhotic NASH and fatty liver disease. But Galectin Therapeutics is the only company with an active Phase 2 clinical trial in NASH cirrhosis, and that is important because of all the things I have told you about how important cirrhosis is. We are the only company focused with a trial there now. So, why do we think we can be effective in NASH cirrhosis? This is a critical underpinning of our program, and I am going to go through that with you, and I think it is important to know why the disease stage is so important.



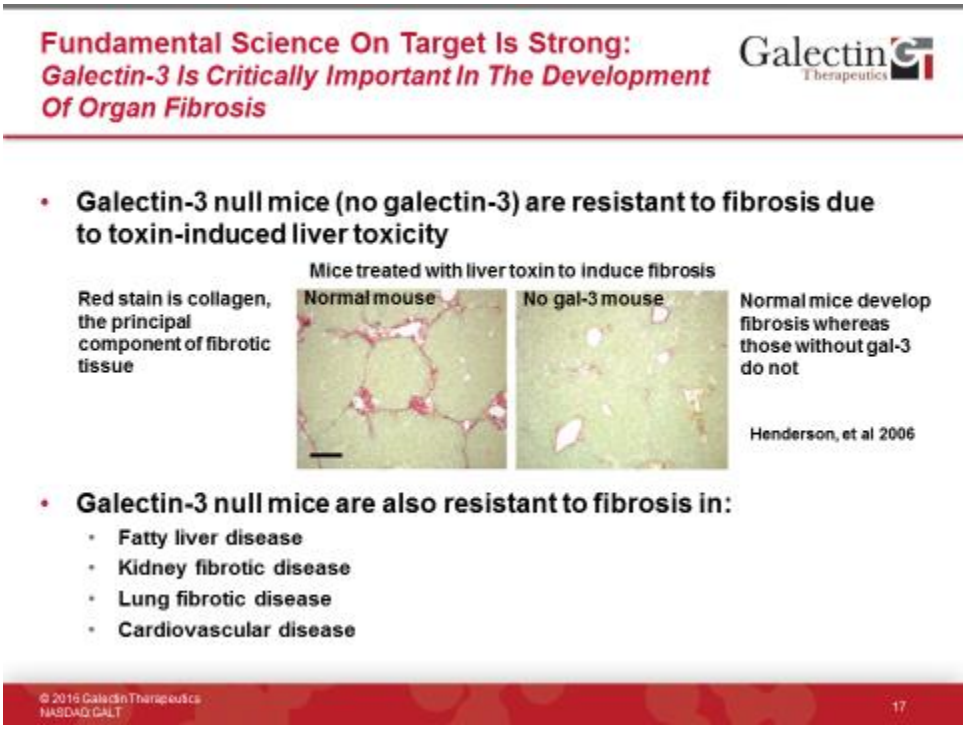
Fatty liver disease progresses slowly and is asymptomatic for a long time. It is difficult to diagnose in early stages, and in early stages can be reversed by lifestyle changes, basically weight loss and exercise.

The majority of fatty liver patients will not reach cirrhosis or have liver-related problems. If the early stages of NASH are targeted for therapy, millions of people will be treated for a liver disorder that was not going to threaten their lives. Now, there may be other benefits of various drugs that you could focus on but not related to liver-related mortality or morbidity.

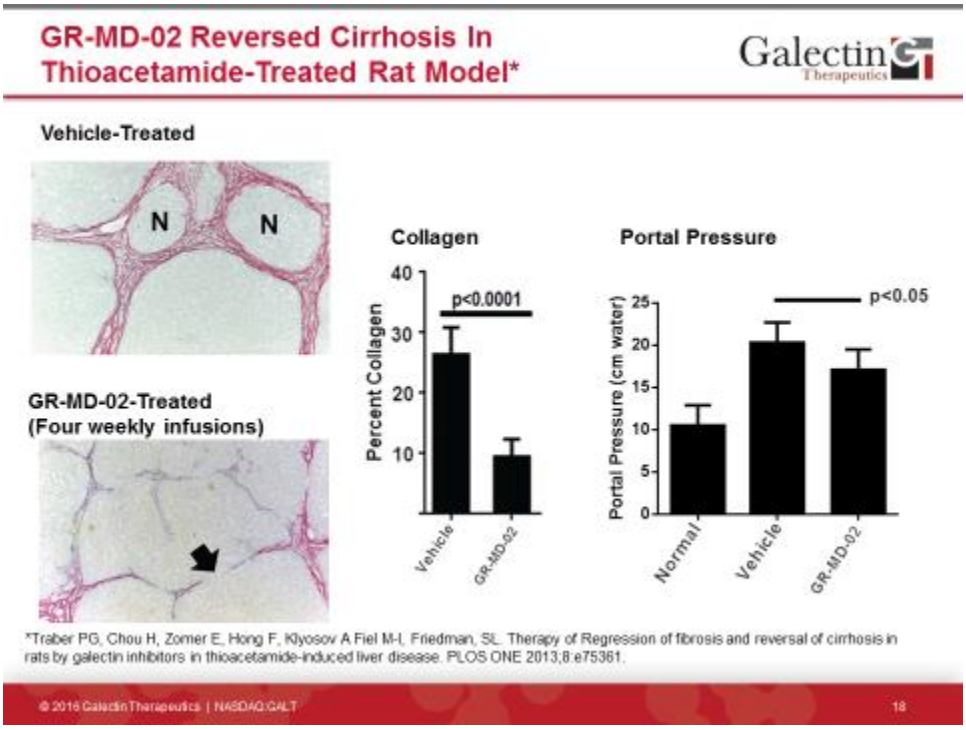


What about targeting cirrhosis? Once NASH progresses to cirrhosis, the patients are at risk for severe complications, liver failure and death. The only current therapy available for NASH cirrhosis is a liver transplant, when clinical progression is severe. Once NASH progresses to cirrhosis, it is not reversible with lifestyle changes.

The goal of our therapy, GR-MD-02, is to reduce fibrosis leading to improved liver function and positively affect outcomes. As I mentioned, we are the only company with a currently active cirrhosis trial.



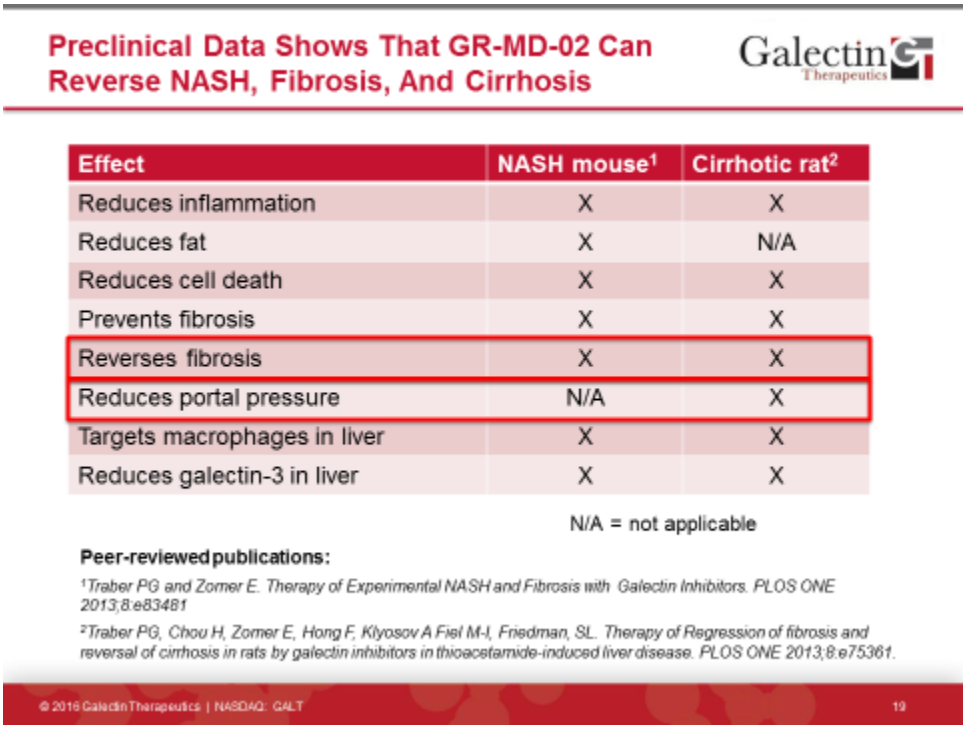
Why do we think this is a good target for us? First of all, the fundamental science is very strong for galectin-3's involvement in fibrosis. In fact, if you remove the galectin-3 gene from animals, they are completely resistant to the development of liver fibrosis. The galectin-3 protein is critically important. You can see that normal mice get fibrotic tissue when treated with a toxin, whereas mice without galectin-3 do not. This has been extended to mice with fatty liver disease, kidney fibrotic disease, lung fibrotic disease, and cardiovascular disease. The galectin-3 gene has been shown with very strong fundamental science to be critical for fibrosis.



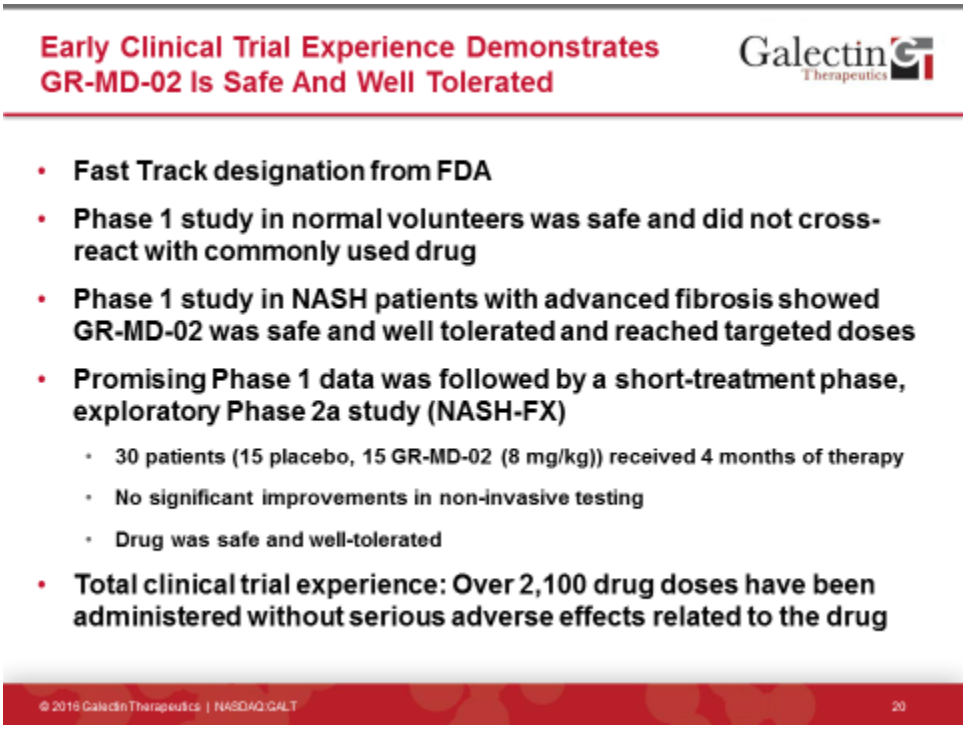
We have done experiments in cirrhotic animals. Rats treated with a toxin get cirrhosis with large bands of fibrotic tissue and nodules; this is cirrhosis. It looks identical to human cirrhosis. After four weekly treatments with GR-MD-02, there was a marked reduction in the fibrosis, thinning of bands and reversal of cirrhosis. You can see it on the quantitative analysis of collagen which is fibrotic tissue, a marked decrease with just four weeks of treatment.

Finally, these animals had an increase in portal pressure which is the blood pressure of the blood going into the liver, and that was also reduced by treatment with GR-MD-02.

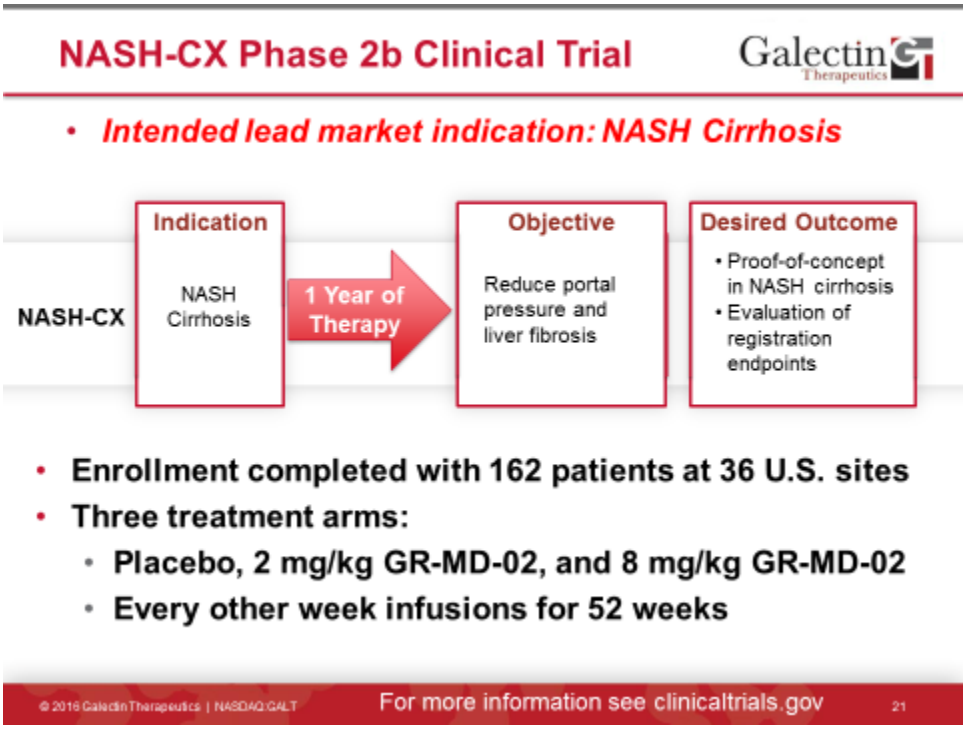
This has all been published in peer review journals and was done by a laboratory of an outstanding investigator in liver disease.



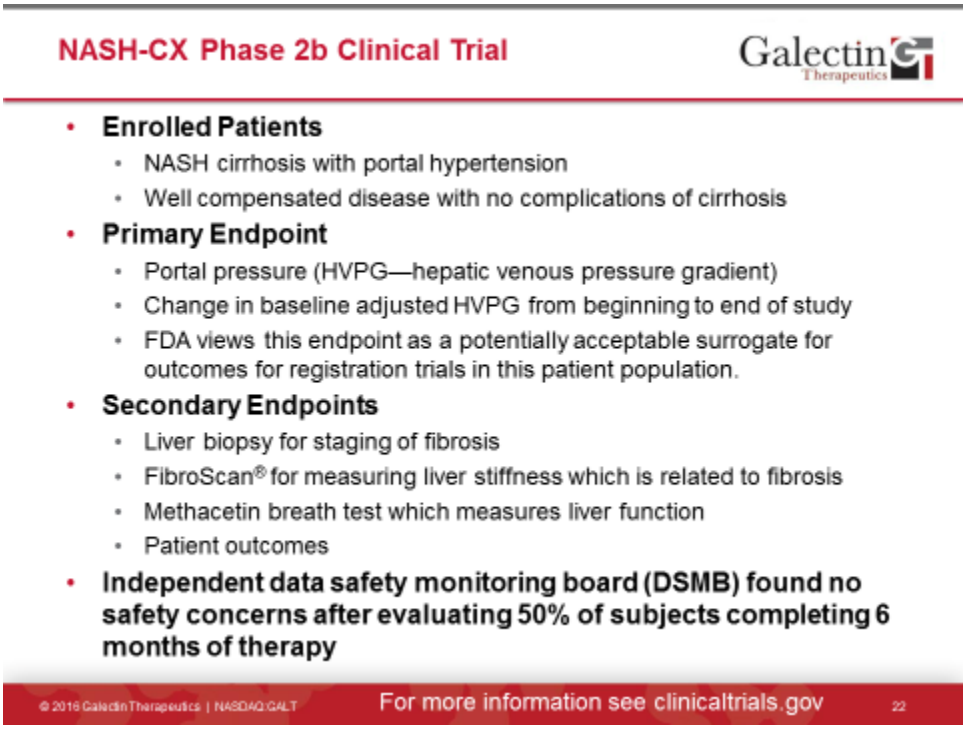
If you look at all of the pre-clinical studies that we have done in mouse models of fatty liver disease and cirrhotic rats, GR-MD-02 has multiple effects reducing inflammation, fat, and cell death, preventing fibrosis but most importantly, reversing fibrosis and reducing portal pressure. It does this by reducing galectin-3 in the liver. The important point is we are targeting NASH cirrhosis, and we have fundamental pre-clinical data to suggest that that is an important and tractable target for our drug.



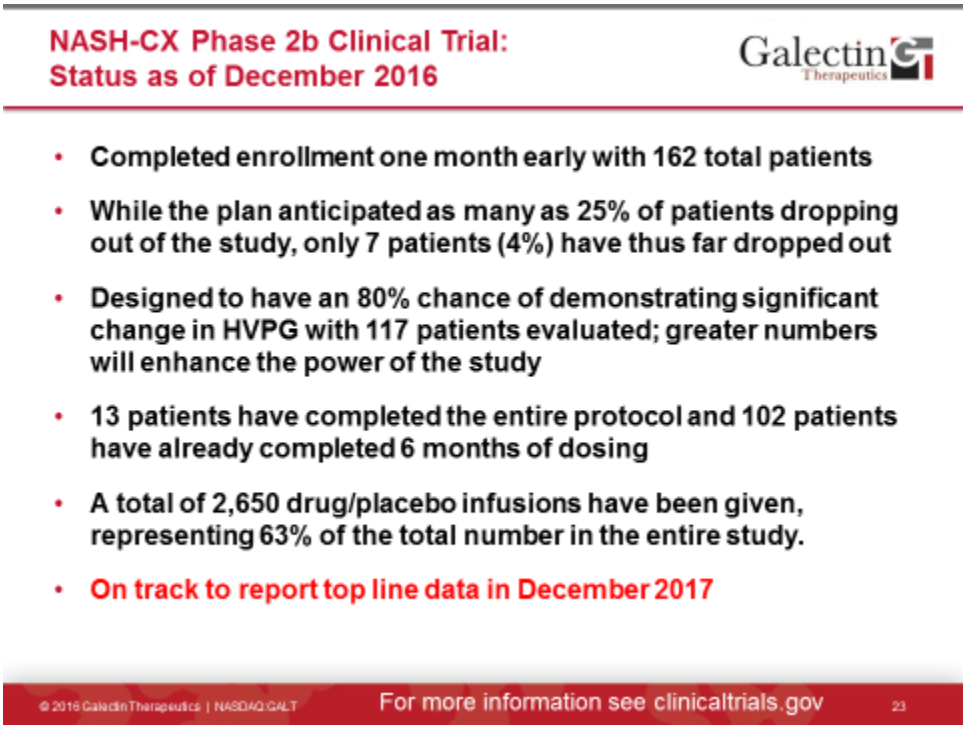
We also have clinical experience with this drug in liver disease, and the early clinical experience demonstrates GR-MD-02 is safe and well tolerated. We received fast track designation from the FDA for this program. A Phase 1 study in normal volunteers was safe, and it showed that the drug does not cross-react with commonly used drugs. We did a Phase I in NASH patients -- fatty liver disease patients -- with advanced fibrosis. It showed that it was safe and well tolerated, and we reached the targeted doses, and we obtained promising data from the Phase I trials on potential therapeutic effects. Therefore, Phase 1 was followed by a short treatment phase exploratory Phase 2a study called the NASH-FX study, where 30 patients were treated, 15 with placebo, 15 with GR-MD-02, for only four months. We found that there wasn't significant improvement in noninvasive testing, but we could not do invasive testing such as liver biopsy in this short study. Again, the drug was safe and well-tolerated. While we followed up on some promising findings in Phase I, the short small exploratory Phase 2 did not show an effect. We now have total clinical experience of over 2100 doses of the drug that have been administered without serious adverse effects related to the drug.



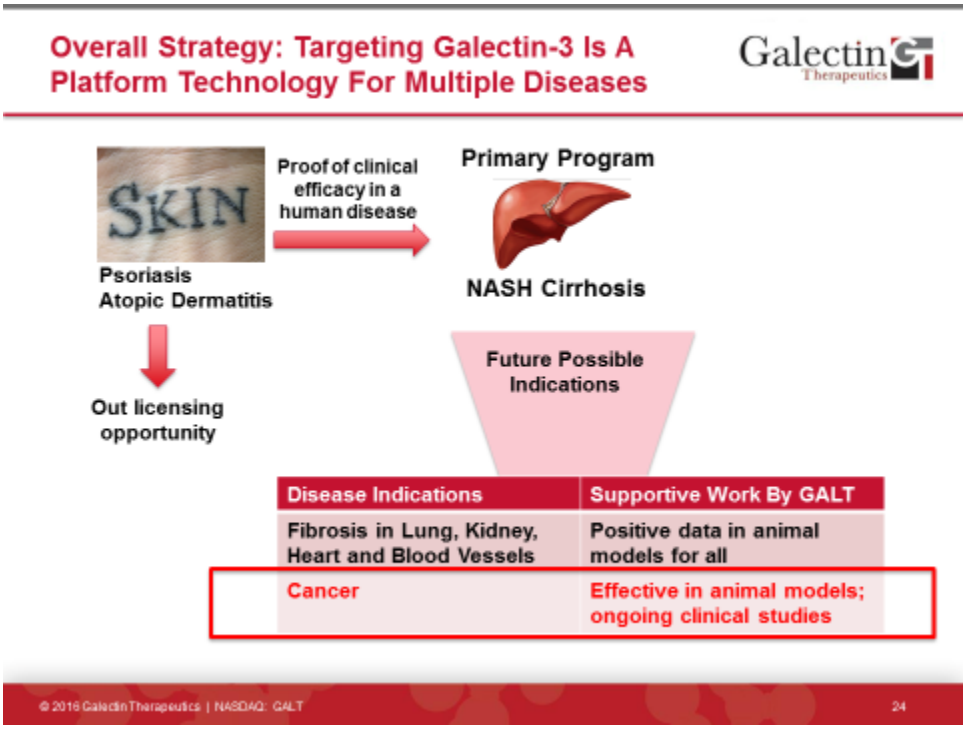
Which brings us to our major Phase 2B clinical trial in NASH cirrhosis, the NASH-CX trial. This is the intended market indication, NASH cirrhosis. All the patients have NASH cirrhosis that is well compensated, in other words no complications of cirrhosis as yet. The trial involves one year of therapy, and the objective is to reduce portal pressure and improve liver fibrosis. This is intended to be a proof of concept in NASH cirrhosis. If successful, it would be the first trial in NASH cirrhosis or any kind of cirrhosis that showed improvement, and we are evaluating registration end points in this trial. Enrollment is complete with 162 patients at 36 sites. We have three treatment arms -- placebo and two doses of drug -- and the patients are given every other week infusions for 52 weeks.



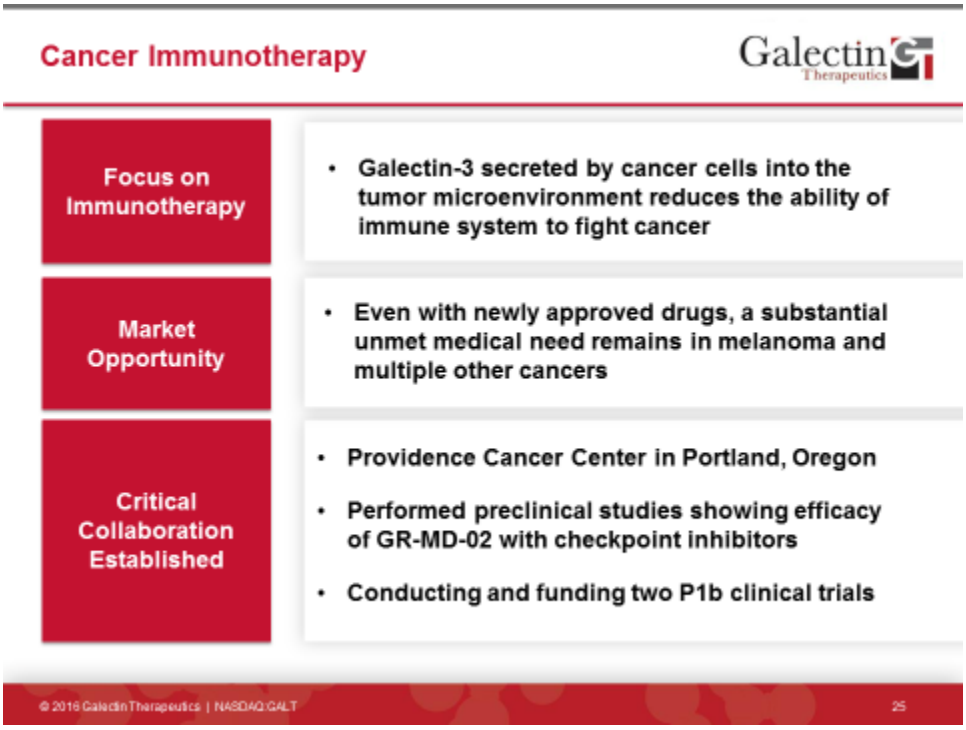
Now let me tell you a little bit more detail about this trial. First of all, all the patients have NASH cirrhosis with portal hypertension. It is well compensated with no complications of cirrhosis. If you will remember that diagram that I showed you, it is at that early cirrhosis stage, where it can show the most likelihood of improvement. The primary endpoint is the portal pressure measured by something called HVPG or hepatic venous pressure gradient. We are looking at a baseline adjusted change from the beginning to the end of the study in HVPG. Importantly, the FDA has indicated that this endpoint is a potentially acceptable surrogate for outcomes for registration trials in this population. It is an important primary endpoint. Secondary endpoints are also important, and we have staging by liver biopsy, stiffness of the liver as measured by FibroScan, Methacetin breath test which measures liver function, and patient outcomes. Importantly, just this past December 9th, the independent data safety monitoring board or DSMB, met to review data. They evaluated the 50% of subjects in the trial who have completed 6 months of therapy. They looked at 80 some patients who completed 6 months of therapy, and they found absolutely no safety concerns. This is a very important milestone for this clinical trial.



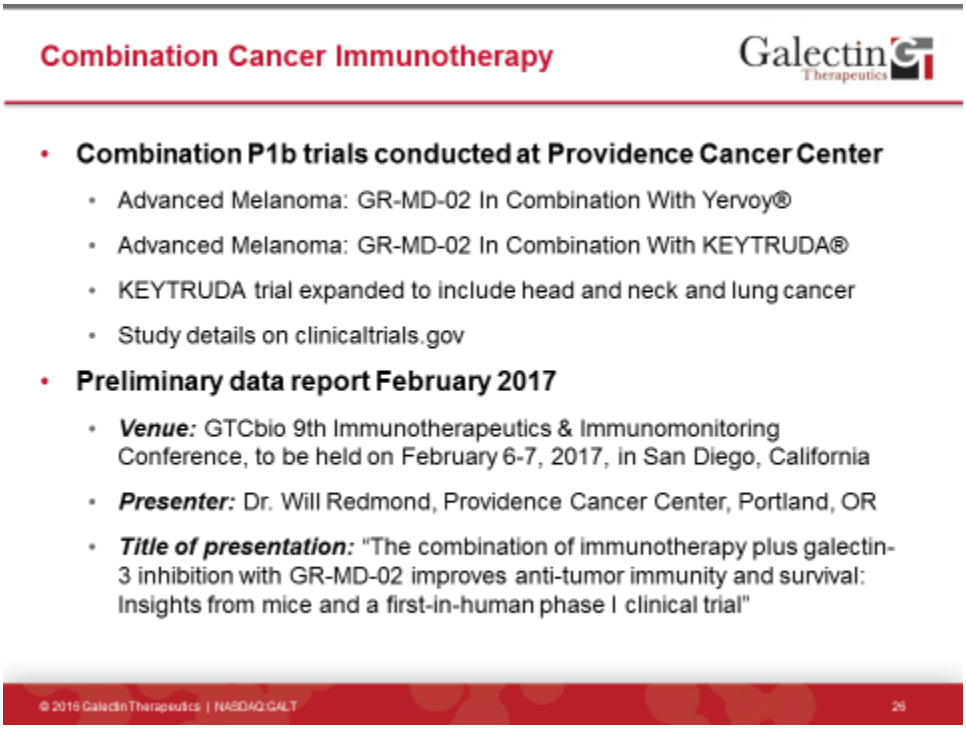
What is the status of this trial as of December of this year? We have completed enrollment and only 4% have dropped out so far. The plan was that as many as 25% may drop out of the study, but we think that the conduct of the study and the safety of the drug is keeping people involved, which is good. We designed the study showing an 80% chance of demonstrating a significant change in HPVG if 117 patients were enrolled. We are likely to have more patients than that because we have 162 enrolled. The number of patients over 117 simply increases the power to be able to show a difference in the study. Thirteen patients have completed the entire protocol, and 102 patients have completed 6 months. Importantly, 2600 drug and placebo infusions have been given which represents 63% of the total number of the entire study. We are on track to report top line data in December of 2017, as we have been advising for some time now.



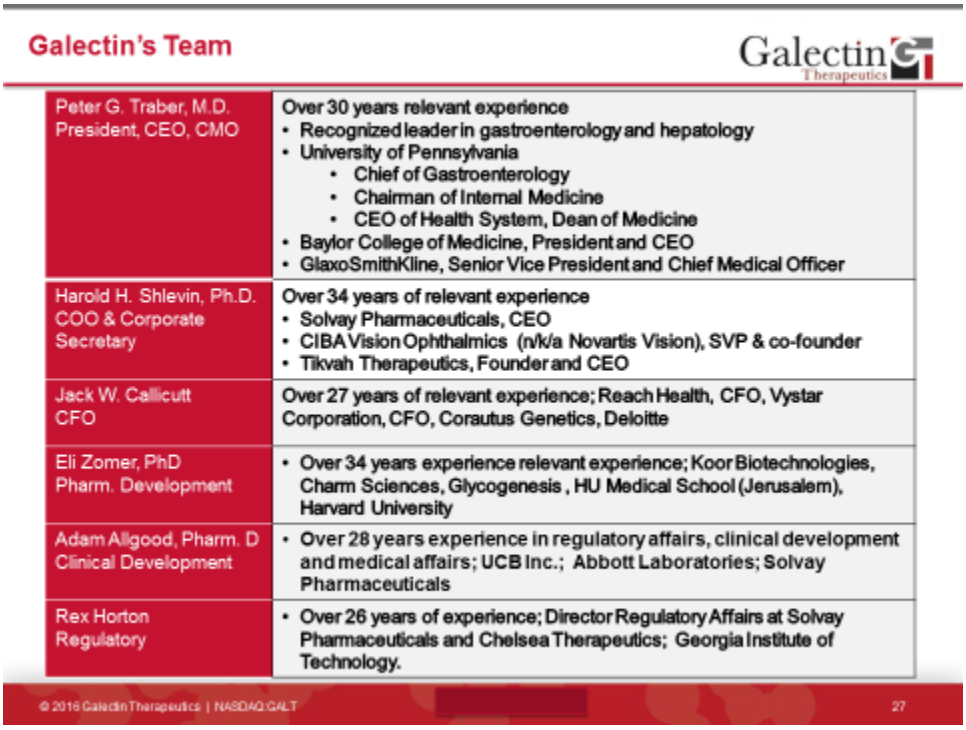
Now, let me come back to these other indications and, as I pointed out on this previous slide, there are other indications, and I want to just talk a little bit about cancer because we do have ongoing clinical work in this area.



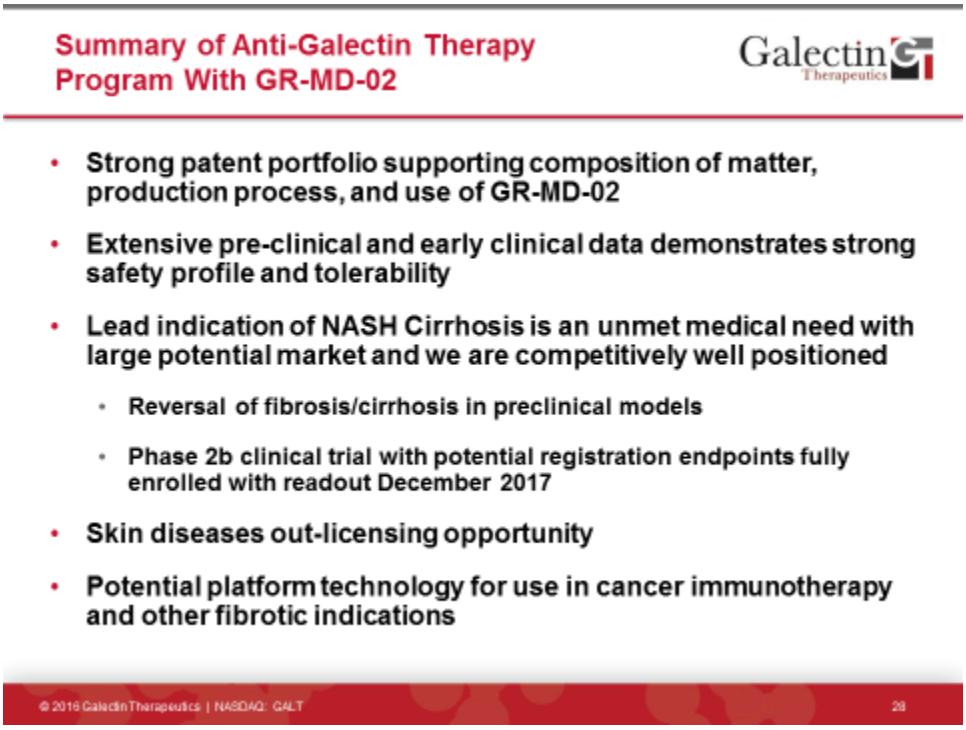
Galectin-3 may be very important in the study of cancer because the vast majority of cancer cells over-express and have more galectin-3 than normal cells. This galectin-3 is secreted into the tumor micro-environment, and it reduces the ability of the immune system to fight the cancer. You can think of it as a little bit of a cloaking device for the tumor to protect it from the immune system. Now, even with newly approved drugs in cancer immunotherapy, there is still a substantial unmet medical need in melanoma as well as other cancers. Some of the news that you hear is about great new therapies. What is not completely clear to everybody is some of those therapies only work in 10%, 20%, or 30% of the patients. They are great new therapies, but that is because it was 1% before and now it's 20%, that sounds pretty good. But that also means there is still substantial room in the other 70% or 80% of people. We are very fortunate that we have a critical collaboration established with the Providence Cancer Center in Portland, Oregon. They have done pre-clinical studies showing that when you combine GR-MD-02 with so-called check point inhibitors which are those newly approved drugs, there's a marked increase in efficacy, and they are conducting and funding two Phase 1B clinical trials.



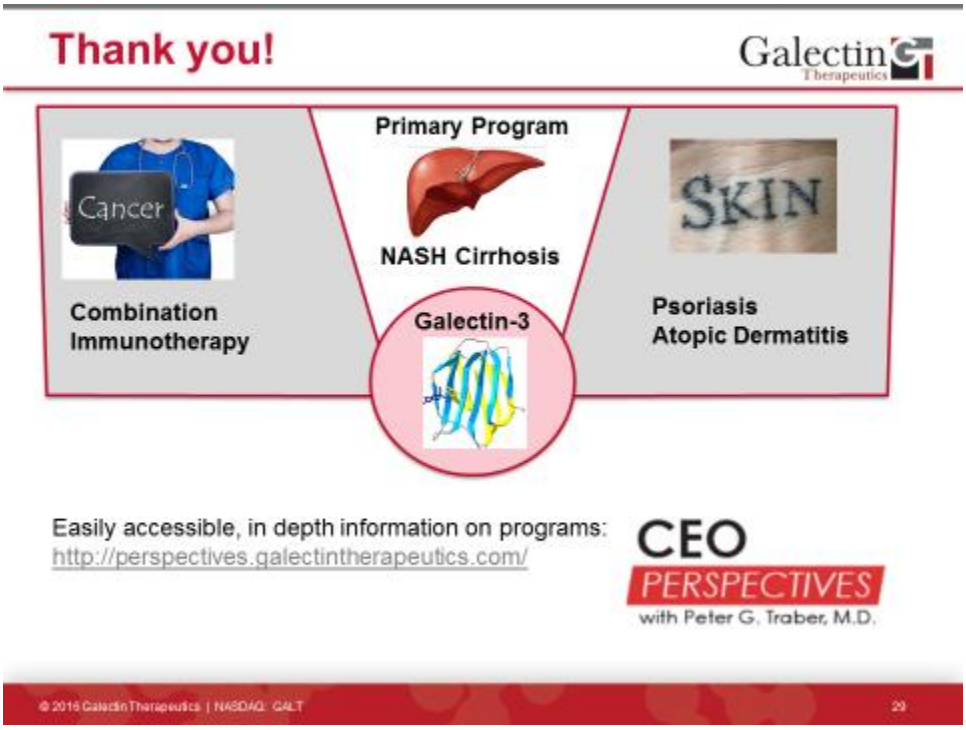
Those clinical trials are in both in advanced melanoma. One is GR-MD-02 in combination with the checkpoint inhibitor Yervoy. The other is in combination with Keytruda. Recently, the Keytruda trial has been expanded to include head and neck and lung cancer. Thedetails on these studies can be found on clinicaltrials.gov. We are anticipating a preliminary data report in February of 2017. Dr. Will Redmond at the Providence Cancer Center will report at the Immunotherapeutics and Immunomonitoring Conference to be held February 6th and 7th in San Diego. He will have a 30-minute talk where he is going to present both pre-clinical data as well as first in human Phase 1 clinical trial experience, and so we will get an update on the patients that have been enrolled in both of these clinical trials at that conference, and we will see how we will proceed forward with the trials based on their presentation.



I want to point out that I am very proud of the team that is involved in doing this work at Galectin Therapeutics. This small but extremely experienced team has put together this program, and we are poised to have important clinical results in 2017.



To summarize what I have said, we have a very strong important patent portfolio supporting GR-MD-02. Extensive pre-clinical and early data demonstrating a strong safety profile and tolerability. Lead indication of NASH cirrhosis is a very big unmet medical need with a large potential market, and we are competitively well positioned in this competitive field. We have shown reversal of fibrosis in pre-clinical models which is the basis for targeting NASH cirrhosis, and we have a Phase 2B clinical trial with potential registration endpoints fully enrolled with a read out in December of 2017. Skin diseases are an out-licensing opportunity for us but also demonstrate that this is an active pharmacological agent that has an important effect on two severe immune-mediated skin diseases. And, it's a potential platform technology for use in cancer immunotherapy and potentially other fibrotic indications.



I want to thank everybody for their attention and thank the stockholders for their confidence and for their investment in the company. I want to point out that there is information on our programs on our website as well as the *CEO Perspectives* blog. With that, I will end this presentation and look forward to communicating with all the stockholders over the upcoming exciting year.