UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

PURSUANT TO SECTION 13 OR 15(d) OF THE
SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): December 1, 2021

180 LIFE SCIENCES CORP.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-38105**	**90-1890354**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

3000 El Camino Real, Bldg. 4, Suite 200 Palo Alto, CA	**94306**
(Address of Principal Executive Offices)	(Zip Code)

Registrant's telephone number, including area code: **(650) 507-0669**

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e 4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	**Trading Symbol(s)**	**Name of each exchange on which registered**
Common Stock, par value $0.0001 per share	ATNF	The NASDAQ Stock Market LLC
Warrants to purchase shares of Common Stock	ATNFW	The NASDAQ Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure

On December 1, 2021, 180 Life Sciences Corp. (the "Company") issued a press release relating to a keynote address presented by Prof. Jagdeep Nanchahal (a consultant to the Company and the Chairman of our Clinical Advisory Board), at the 2021 International Dupuytren Symposium, held on December 1, 2021, including top line data from the Company's phase 2b clinical trial associated with the use of adalimumab as a potential therapeutic target to help treat Dupuytren's disease.

A copy of the press release is furnished hereto as Exhibit 99.1.

A transcript of Prof. Nanchahal's presentation is furnished herewith as Exhibit 99.2.

The information in Item 7.01 of this Form 8-K and Exhibits 99.1, and 99.2, attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act") or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such a filing. The furnishing of this Report is not intended to constitute a determination by the Company that the information is material or that the dissemination of the information is required by Regulation FD.

The press release furnished as Exhibit 99.1 to this Current Report on Form 8-K and the transcript furnished as Exhibit 99.2, contain forward-looking statements within the safe harbor provisions under The Private Securities Litigation Reform Act of 1995, and, as such, may involve known and unknown risks, uncertainties and assumptions. These forward-looking statements relate to the Company's current expectations and are subject to limitations and qualifications set forth in the press release and transcript, and as well as in the Company's other filings with the Securities and Exchange Commission, including, without limitation, that actual events and/or results may differ materially from those projected in such forward-looking statements. These statements also involve known and unknown risks, which may cause the results of the Company, its divisions and concepts to be materially different than those expressed or implied in such statements. Accordingly, readers should not place undue reliance on any forward-looking statements. Forward-looking statements may include comments as to the Company's beliefs and expectations as to future financial performance, events and trends affecting its business and are necessarily subject to uncertainties, many of which are outside the Company's control. More information on potential factors that could affect the Company's financial results is included from time to time in the "Forward-Looking Statements," "Risk Factors" and "Management's Discussion and Analysis of Financial Condition and Results of Operations" sections of the Company's periodic and current filings with the SEC, including the Form 10-Qs and Form 10-Ks, filed with the SEC and available at

www.sec.gov. Forward-looking statements speak only as of the date they are made. The Company undertakes no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise that occur after that date, except as otherwise provided by law.

Disclaimer

The information contained in the transcript furnished as Exhibit 99.2 is a textual representation of a video recording of the presentation described above, and while efforts are made to provide an accurate transcription, there may be material errors, omissions or inaccuracies in the reporting of the substance of the recording. The Company does not assume any responsibility for any investment or other decisions made based upon the information provided in this transcript. Users are advised to review the video recording and the Company's SEC filings before making any investment or other decisions. An archived recording of the presentation is accessible through the International Dupuytren website at https://dupuytrensymposium.org/scientific-program.

The link to the website above is provided for informational purposes only and we make no representations regarding such website, the information thereon, or any links accessible therefrom, and do not desire to incorporate any of the information on, or accessible through, such website into this report.

Item 9.01. Financial Statements and Exhibits

(d) Exhibits.

Exhibit No.	Description
99.1*	Press Release dated December 1, 2021
99.2*	Transcript of presentation presented by Prof. Jagdeep Nanchahal, at the 2021 International Dupuytren Symposium, held on December 1, 2021
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

* Furnished herewith.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: December 1, 2021

180 LIFE SCIENCES CORP.

By: */s/ James N. Woody, M.D., Ph.D.*
Name: James N. Woody, M.D., Ph.D.
Title: Chief Executive Officer

Exhibit 99.1

180 Life Sciences Corp. Co-Founder Prof. Jagdeep Nanchahal Presents Keynote Address at the 2021 International Dupuytren Symposium, including top line data from the phase 2b clinical trial

'Cellular and Molecular Profiling of Dupuytren's Disease: from the Lab to the Clinic' Presented Wednesday, December 1, 2021, at 3pm EST

PALO ALTO, Calif., December 1, 2021 (GLOBE NEWSWIRE) -- 180 Life Sciences Corp. (NASDAQ: ATNF) ("180 Life Sciences" or the "Company"), a clinical-stage biotechnology company focused on the development of novel drugs that fulfill unmet needs in inflammatory diseases, fibrosis and pain, today announced that Professor Jagdeep Nanchahal from the University of Oxford, a co-founder of 180 Life Sciences, presented a keynote address entitled 'Re-purposing anti-TNF for Dupuytren's Disease' at the 2021 International Dupuytren Symposium on Wednesday, December 1, 2021.

As part of the keynote address, Professor Nanchahal described how his team unraveled the molecular mechanisms underlying the pathogenesis of Dupuytren's disease leading to the identification of Tumor Necrosis Factor (TNF) as a potential therapeutic target and clinical trials he led to identify the optimal dose and formulation of adalimumab, an anti-TNF biologic.

At the keynote, he also disclosed the top line data from the phase 2b clinical trial, for patients with early-stage disease, which met the primary end point of nodule hardness and the secondary end point of nodule size on ultrasound scan with statistically significant differences. There were no related severe adverse events. The full results have been submitted for publication in a peer-reviewed journal and will be disclosed on publication.

Additional information regarding Prof. Nanchahal's address, found in the Current Report on Form 8-K which 180 Life Sciences filed today with the Securities and Exchange Commission.

The below is a link to the video presentation of Prof. Nanchahal's address which will be accessible through the International Dupuytren website (https://dupuytrensymposium.org/scientific-program/).

About 180 Life Sciences Corp.

180 Life Sciences Corp. is a clinical-stage biotechnology company focused on the development of novel drugs that fulfill unmet needs in inflammatory diseases, fibrosis and pain by leveraging the combined expertise of luminaries in therapeutics from Oxford University, the Hebrew University and Stanford University. 180 Life Sciences is leading the research into solving one of the world's biggest drivers of disease – inflammation. The Company is driving groundbreaking studies into clinical programs, which are seeking to develop novel drugs addressing separate areas of inflammation for which there are no effective therapies. The Company's primary platform is a novel program to treat fibrosis using anti-TNF (tumor necrosis factor).

Forward-Looking Statements

This press release includes "forward-looking statements", including information about management's view of the Company's future expectations, plans and prospects, within the safe harbor provisions under The Private Securities Litigation Reform Act of 1995 (the "Act"). Words such as "expect," "estimate," "project," "budget," "forecast," "anticipate," "intend," "plan," "may," "will," "could," "should," "believes," "predicts," "potential," "continue" and similar expressions are intended to identify such forward-looking statements. These forward-looking statements involve significant risks and uncertainties that could cause the actual results to differ materially from the expected results and, consequently, you should not rely on these forward-looking statements as predictions of future events. These forward-looking statements and factors that may cause such differences include, without limitation, statements relating to expectations regarding the capitalization, resources, and funding of the Company; statements regarding adalimumab's potential as a treatment for Dupuytren's disease; that the top-line data 180 Life Sciences has reported is based on preliminary analysis of key efficacy and safety data, and such data may change following a more comprehensive review of the data related to the clinical trial and such top-line data may not accurately reflect the complete results of the trial expectations with respect to future performance, growth and anticipated acquisitions; the continued listing of the Company on The NASDAQ Stock Market; expectations regarding the capitalization, resources and ownership structure of the Company; expectations with respect to future performance, growth and anticipated acquisitions; the ability of the Company to execute its plans to develop and market new drug products and the timing and costs of these development programs; estimates of the size of the markets for its potential drug products; potential litigation involving the Company or the validity or enforceability of the intellectual property of the Company; global economic conditions; geopolitical events and regulatory changes; the expectations, development plans and anticipated timelines for the Company's drug candidates, pipeline and programs, including collaborations with third parties; access to additional financing, and the potential lack of such financing; and the Company's ability to raise funding in the future and the terms of such funding. These risk factors and others are included from time to time in documents the Company files with the Securities and Exchange Commission, including, but not limited to, its Form 10-Ks, Form 10-Qs and Form 8-Ks. These reports and filings are available at www.sec.gov. All subsequent written and oral forward-looking statements concerning the Company, the studies described herein or other matters and attributable to the Company or any person acting on its behalf are expressly qualified in their entirety by the cautionary statements above. Readers are cautioned not to place undue reliance upon any forward-looking statements, which speak only as of the date made, including the forward-looking statements included in this press release, which are made only as of the date hereof. The Company cannot guarantee future results, levels of activity, performance or achievements. Accordingly, you should not place undue reliance on these forward-looking statements. The Company does not undertake or accept any obligation or undertaking to release publicly any updates or revisions to any forward-looking statement to reflect any change in its expectations or any change in events, conditions or circumstances on which any such statement is based, except as otherwise provided by law.

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Transcript of keynote address presented by Prof. Jagdeep Nanchahal, at the 2021 International Dupuytren Symposium, held on December 1, 2021

Speaker: Prof. Jagdeep Nanchahal

I would like to start by thanking the organizers for inviting me to present this keynote lecture I've also taken the liberty of slightly amending the title of my presentation as I would like to take this opportunity to present my journey of the work that spans a period of over 14 years of how we dissected the cellular and molecular profile of Dupuytren's disease, identified key signaling mechanisms and how I translated these findings through to Phase 2 clinical trials

Dupuytren's disease is incredibly common and this is the real issue, that treatment options are currently limited to late stage disease. We've heard over the last few weeks at this Symposium we can cut it out or we can disrupt it with needles or collagenase, but this isn't what patients want. They know when their Dupuytren's disease is at an early stage and currently there's no validated approved treatment. In our review here in 2016 and we assessed the available literature and found that in fact there are no clinical trials with control groups and blinding of the participants and the assessors for some of the current treatment options that are used, such as intralesional steroid injection or radiotherapy.

What I just described is rather reminiscent of we used to see patients with rheumatoid arthritis back in the 1990s when I was a surgical trainee. Patients used to present with florid synovitis, which we as surgeons used to excise and invariably reoccurred, and some clinicians suggested that patient should even be treated with radiotherapy. It was only when this group back in 1989 defined TNF as a potential therapeutic target that totally changed that landscape of how these patients were managed. They went on to get the Lasker award and Anti TNF has essentially gone on to become the standard of care together with a whole host of other monoclonal antibodies such as IL-6R etc.

Today I'm going to describe how my group is dissected out the cellular and molecular profile of Dupuytren's disease, the underlying cellular mechanisms. How the various subgroups of cells maintains the disease ecosystem characterized by chronic low-grade inflammation, how we identified TNF as a potential therapeutic target, the data from our dose ranging Phase 2A Clinical trial which has been published. I'll also share with you the top line data of the Phase 2B Clinical Trial in patients with early stage Dupuytren's disease.

The early stage of DD is represented by these highly cellular nodules. Our single cell RNA sequencing and CyTOF studies show that they comprise a mixed population of fibroblasts, myofibroblasts, endothelial cells, immune cells and pericytes.

Let me take you through these each in turn. First the fibroblasts. These are not a homogeneous population and there at least four stop sets. I draw your attention in particular to this ICAM1+ IL6 high which are relatively small population especially enriched within the nodules and they play a key role in attracting the immune cells to the site of disease. The myofibroblasts are also not homogeneous. They range from this ACTA2 low group, to intermediate to a CD82 high population, which is highly contractile as shown here in this single cell contractility assay. It is these intermediate and CD82 high cells that are mainly responsible for matrix production and remodeling. The cycling population don't contribute very much to matrix production, instead they do exactly what their name suggests, they maintain the pool of myofibroblasts. We also looked at the different immune cells and as you can see there are several different subsets macrophages, dendritic cells, T cells and not apparent in this sequencing study, because the numbers are too small, we also see very low numbers for mast cells, which are very important.

With freshly disaggregated cells from nodules we see a whole array of cytokines being produced. I don't want to take you through all of these. However, I'd like to draw your attention in particular to TGFb, which is the archetype of cytokine associated with fibrosis. you can see about 300pg/ml, relatively large amounts of IL-6, quite small amounts of TNF, around 50pg/ml and even lower amounts of IL-33. We tested the effects of many of these in functional assays and I will not take you through all of these, but let's look at a couple of key ones.

I will start by defining the effects of TNF in the various cell populations. This was particularly interesting because what we found was that very low levels, around 100pg/ml with the dose broken down here somewhere between 50 and 100pg/ml, only palmar fibroblasts from Dupuytren's patients became much more contractile, but at higher doses it is inhibitory and interestingly if you take non-palmar cells from the same Dupuytren's patients they didn't respond in the same way and neither did palmar cells for normal individuals; in fact they become less contractile but at much higher concentrations of TNF. If you take myofibroblasts from Dupuytren's patients and expose them to the anti TNFs, here the data is showing the data for the approved anti TNF agents that are used for other conditions such as rheumatoid arthritis, you see downregulation contractility which is accompanied by down regulation of aSMA protein and COL1messenger RNA.

The results of TGFb are very different. This cytokine converts all cell types, irrespective of their origin, into much more contractile myofibroblast cells but at much higher concentrations than we see physiologically. You'll recall those freshly disaggregated cells from the nodules TGF b levels are around 300pg/ml. We begin to see effects now at about 1,000 and even 10,000pg/ml. It is acting indiscriminately and predictably when you inhibit TGFb, you see a downregulation of myofibroblast contractility as well as downregulation of aSMA protein and COL1 expression.

I've shown you data that inhibition of either TNF or TGFb is effective in down regulating the myofibroblast phenotype. So why select TNF when in freshly dissected nodular cells we see around 50 picograms per ml of TNF and around 300 picograms per ml of TGFb in the supernatant. The cells are often sub-cultured up to passage 5 or 6 and already at passage 2 we see the TNF concentrations falling off because the immune cells have largely disappeared. Because of autocrine secretion by the stromal cells we now seeing a huge upregulation of TGFb production. So this suggests that TGF levels seen here later passaged cells is a culture artifact and this goes some way to explaining the failure of targeting TGF in multiple late Phase clinical trials.

My colleague, Dominic Furniss in Oxford and Paul Werker from Groningen have shown the key role of Wnt signaling pathways in Dupuytren's disease through their genome wide association studies. We went on the show that TNF only in palmar fibroblast from Dupuytren's patients acts via the Wnt signaling pathway to upregulate genes such COL1 and aSMA typically associated with fibrosis. And now we have both molecular and genetic data supporting TNF as a potential therapeutic target. We know that the genetics of Dupuytren's disease is incredibly important as a hereditability about 80% but Dupuytren's disease is restricted to the palm of genetically susceptible individuals so this suggests that epigenetic regulation must play a key role and indeed it does. Histone acetylation is associated with increased gene expression and what we have shown is that histone acetylation in myofibroblasts is crucial in maintaining their phenotype. Conversely methylation suppresses gene expression and our most recent data show that this mechanism is important in regulating cytokine expression by those immune regulatory fibroblasts through an intermediatory molecule called PLAG2G2A. So suppression of these genes leads to increased expression CCL2 & CCL7, which in turn recruit the immune cells and these produced the TNF that drives the myofibroblast phenotype.

Dupuytren's disease persists and develops over a period of many decades. Typically inflammation in humans occurs and then resolves over a period of weeks and months. What we see here in Dupuytren's disease is the inflammation persisting over very long period. What we found is that those immune cells produce TNF which drives the development of myofibroblasts and these in turn produce low levels of cytokine called IL-33, which in turn acts on the immune cells and hence we get this vicious cycle whereby the information persists over very long period.

Our most recent data has showed that that the various cell types that I've described are not randomly distributed through the nodule but in fact compartmentalized so you have these immune regulatory fibroblasts which attract the immune cells which in turn produce TNF which drives the myofibroblasts phenotype, but these immune regulatory fibroblasts are maintained by platelet derived growth factor produced by the fibrotic endothelium and there is this relative segregation of the myofibroblast compartment from the remainder. We see a very similar pattern here in lung fibrosis in patients with a idiopathic pulmonic fibrosis. So segregated myofibroblasts and then these immune regulatory fibroblasts and blood vessels around the myofibroblast foci.

Having identified TNF as a potential therapeutic target I applied to the Wellcome Trust and Department of Health for funding to undertake a Phase 2A followed by a Phase 2B randomized clinical trial. For Phase 2A if we were to use clinical outcome measures, we'd need relatively large number of patients in each group. So instead I chose an experimental medicine design including patients were already scheduled for surgery but who had large prominent nodules in the palm of their hand that were to have been excised at the time of surgery. We injected those nodules with different doses of anti-TNF or an equivalent volume of placebo in a blinded manner collected the tissue after surgery and then analyzed it in the lab for various markers which I will show you in moment.

Having identified the optimal dose of the anti-TNF we then preceded to a Phase 2B clinical trial where we were treated patients with early stage disease and prominent nodule with a clear history of activity within that nodule and patients were randomized to receive either that optimal dose of anti-TNF or an equivalent volume of saline. Now adalimumab as you know in patients with rheumatoid arthritis is administered every two weeks. However our patients would not tolerate 2 injections every two weeks so we did end user survey. We asked patients with both early and late stage disease how many injections a year would they accept to control their Dupuytren's disease. Almost all said one injection a year the majority said two and three and once we get to about four the positive responses were beginning to fall off. On that basis we designed the trial such that patients would receive 4 injections of the TNF and an equivalent volume of placebo every three months for a year and then we followed them for a total of 18 months. Dupuytren's diseases is a localized low grade inflammatory disease I hypothesized that if the anti-TNF is going to be effective it has to be delivered locally into the nodule at high concentrations. The nodules can be identified clinically and here you can see them with ultrasound scan. What we found with our ex vivo studies that around 0.3 to 0.5 ml remains constrained within the nodule, and after that it just begins to spill out.

So this is the design of the first Phase 2A Clinical Trial. These are patients with nodules that are going to be excised and two weeks before their surgery we injected them with different doses of adalimumab so we first injected them with 15mg in 0.3 ml or an equivalent volume of saline, next 35mg in 0.7 ml or equivalent volume of saline and it was at this stage that I notice that the 0.7 ml is not remaining constrained within the nodule as we've seen in the ex vivo studies but in fact was spilling out into the subcutaneous space.

Now I had hypnotized that we would need high local concentrations of anti-TNF if it were to be effective because this is a localized fibrotic disease and by coincidence the manufacturer that point brought in a new formulation much more concentrated at 40mg in 0.4 ml. The great thing about this formulation is that is was free of excipients such as citrate which it should be associated with lower pain on injection. So we submitted a major protocol amendment incorporating additional dose forward before we had analyzed and unblinded data so we now had three different doses 15, 35 and 40mg in different volumes. Having collected the tissue we then analyzed it in a blinded manner 2 weeks following the injection of the anti-TNF or equivalent volume of placebo. We found was that only the 40mg in 0.4 ml led to a downregulation in aSMA protein concentration as well as collagen I protein concentration. Contrast that with the 35 milligrams - for very similar amounts of adalimumab and half the concentration in this had no effect on either aSMA or collagen I. so this study allowed us to identify the optimal dose and formulation and we can then take forward to the next stage of our study.

We published the results of our Phase 2A Clinical Trial in EBiomedicine in 2018. Having identified the optimal dose and formulation about adalimumab to use for local administration in patients with Dupuytren's disease, we preceded to a randomized double blind placebo controlled trial. We recruited patients with early stage Dupuytren's disease who did not have established flexion deformities that would be treated surgically but did have a prominent nodule with a clear recent history of progression. The first question we asked was how often to inject the nodule. Adalimumab is administered every two weeks for patients with inflammatory arthritis we did not think that two weekly injections which can be painful will be tolerated therefore we conducted an end user survey we asked patients with early and patients with late stage disease how many injections will be acceptable every year to control the progression of Dupuytren's disease. All agreed one, most said 2 or 3 but after four the acceptability started to fall off. Therefore, we selected 4 injections at three monthly intervals. The next question was which outcome measure to use. Correction of established flection deformities has been used in studies for patients with late-stage disease for example following treatment with collagenase. However using the converse, prevention of flexion deformity poses difficulties. The available data suggests that without treatment 20% of patients with early-stage disease progressed to develop flection deformities over seven years and about 35% by 18 years. These long timescales would mean that we would have to follow patients for ten years or more, which is not feasible in the context of a clinical trial.

Nodule hardness has been uses to access efficacy for example in patients receiving intralesional corticosteroid injections. However in these studies unblinded clinicians assessed clinically whether the is the nodule softer. We wanted to quantitatively measure module hardness. Our literature search revealed that a durometer has been found to be reliable and sensitive for assessing skin hardness in patients with scleroderma, lipodermatosclerosis and scars. Therefore, we use the same durometer. The next challenge we faced was how to calculate sample size. In a pilot study independent we assessed the hardness of nodules in 25 patients in early-stage disease and the corresponding area in the palm of age, sex matched controls. This gave us the difference and standard deviation to calculate sample size. Our pilot data also showed that we could reliably image the nodule using ultrasound skin. Therefore, we measured nodule size using ultrasound scan as a secondary outcome measures as well as various clinical outcomes including patient reported measures.

At this stage I can share with you the top line data of this phase 2B clinical trial. We met the primary endpoint of nodule hardness and the secondary endpoint of nodule size using ultrasound scan, with statistically significant differences. The full results have been submitted for publication in a peer reviewed journal and will be disclosed on publication.

In summary, I have shown you our data that the nodules of early stage Dupuytren's disease comprise a complex ecosystem of vascular and immune cells, fibroblasts and myofibroblasts. There's also clear division of Labor between each of the cell types. The individual cells secrete growth factors to sustain the immune regulatory fibroblasts. These fibroblasts in turn recruit the immune cells. The immune cells secrete cytokines that promotes myofibroblast development and there are distinct populations of myofibroblasts, a small cycling pool and others that are mainly responsible for matrix production and contractility.

A complex cross talk between the stromal and immune cells maintains the chronic low grade localized inflammation characteristic of early stage Dupuytren's disease. Epigenetic regulation is also very important in the modulation of activities of the immune regulatory fibroblasts and myofibroblasts.

A detailed understanding of the mechanisms underlying Dupuytren's disease has allowed us to identify TNF as a potential therapeutic target. We identified the optimal dose and formulation about adalimumab in our Phase 2A clinical trial and I described the top line data of our Phase 2B clinical trial, where we met the primary endpoint of nodule hardness and the secondary endpoint of nodular size on ultrasound scans, both with statistically significant differences. There were no related serious adverse events in the Phase 2B trial.

I would like to finish by acknowledging my laboratory team as well others not shown here. The Clinical Trials team, my colleagues in the Oxford Clinical Trials Research Unit and many collaborators, Dominic Davidson and Paul Werker, in particularly who recruited patients to the RIDD trial as well as surgical colleagues around the country who sent us patient samples, the patients who donated tissue and I am particularly indebted to the RIDD trial participants who continued with all of their follow-ups, including between

the various lockdowns in 2020. None of this work would be possible without the generous supporters of various Funding Agencies shown here, the Department of Health and in particular The Wellcome Trust for supporting the RIDD trial. I've received industry support for the laboratory studies and also for purchase of the drug for the RIDD trial.

Please note: 180 Life Sciences Corp. used its best efforts to provide the foregoing transcript based on the video and audio associated with the presentation, but there cannot be assurance there are no errors, omissions, or inaccuracies.