Slide 1 (00:00)

Michael Shleifer: Hello. Thank you for joining us. I'm Michael Shleifer, CEO and chairman of Biotech Acquisition Corporation and co-founder and managing partner of SPRIM Global Investments. I'm joined by Dr. Wendye Robbins, president and CEO of Blade Therapeutics, to discuss a proposed merger of Blade Therapeutics with Biotech Acquisition Corporation, also known as BAC.

We're very excited about this proposed business combination and are confident in the capability of Blade to create value in the near and long term.

Slide 2 (00:28)

Please note that this presentation is neither an offering of securities nor the solicitation of a proxy vote. The information discussed in this recording is qualified in its entirety by the information in BAC's Current Report on Form 8-K that was filed in connection with the proposed transaction, and which may be accessed on the SEC's website. We urge the shareholders of BAC to read the Form 8-K and BAC's other SEC filings in connection with the proposed transaction carefully because they contain important information about the proposed transaction and Blade's business.

Additionally, during this presentation, we will make certain forward-looking statements that reflect our current views related to our future financial performance, future events, and industry and market conditions; as well as forward-looking statements related to the business combination, such as the anticipated timing, proceeds and benefits of the transaction, and statements about the potential attributes and benefits of Blade's candidate therapies and the format and timing of Blade's development activities and clinical trials.

Slide 3 (01:29)

These forward-looking statements are subject to risks, uncertainties and assumptions that could cause actual results to differ materially from such forward-looking statements. We strongly encourage you to review the Form 8-K that was filed in connection with the proposed transaction, along with the press release and presentation included as exhibits to the Form 8-K, and the information that BAC files with the SEC, particularly those described in the risk factors sections of BAC's filings. BAC does not assume any obligation to update any forward-looking statements, except as required by law.

We note that the statements made regarding expected cash and equity ownership following the closing of the proposed transaction do not take into account any possible redemptions by existing BAC shareholders prior to the closing of the business combination.

Slide 4 (02:18)

Biotech Acquisition Company, or BAC, is a SPAC that completed its initial public offering in January of this year, through which it raised 230 million dollars. BAC is an affiliate of SPRIM, a global healthcare consulting and investment firm with more than 20 years of industry experience and operations in 17 countries. SPRIM's focus is to invest in seed and early-stage biotech and digital health companies. At SPRIM, we believe that the formation of BAC was a logical way to extend the SPRIM team's scientific and clinical expertise to later-stage companies.

BAC's message to investors at IPO was that it is leveraging deep industry expertise to bring an important biotechnology opportunity to investors. We believe we have done this with the proposed business combination with Blade.

Slide 5 (03:10)

Blade is researching novel biological pathways foundational to cell- and tissue-damage responses. They are focused on developing potential disease-modifying therapeutics in fibrosis and neurodegeneration.

Blade's lead program, cudetaxestat, is a non-competitive autotaxin inhibitor with differentiated preclinical and biochemical characteristics supporting a treatment profile in fibrosis. Blade also has a neurodegeneration capability targeting the role of dimeric calpains in poly-Q conditions. Blade has what we believe is a differentiated pipeline of product candidates with multiple anticipated milestones.

Again, we're very enthusiastic about this proposed merger. I'm pleased to turn the presentation over to Wendye, who will serve as CEO for the new company. She will take you through the details of the planned path ahead. Wendye?

(04:01) Wendye Robbins: Thank you, Michael.

The team at Blade is enthusiastic, as well. We believe that Blade has industry leading capabilities in fibrosis and neurodegeneration. Our cutting-edge science has the potential to transform currently progressive, terminal fibrotic and neurodegenerative diseases into survivable conditions. These debilitating diseases currently afflict and eventually kill, unfortunately, millions of people worldwide.

Before diving into the science, I'll begin with the financing. Our clinical-stage pipeline will be fueled by what we anticipate being a strong cash position post-transaction. This slide illustrates the financial details of the pending transaction, including the enterprise value, the SPAC capital and the PIPE investment. After paying anticipated transaction expenses and assuming no redemptions by BAC's existing shareholders, we anticipate approximately 255 million dollars in cash to be available between BAC's existing cash and the new proceeds from our PIPE, which will come in at approximately 24 million dollars, and before estimated transaction expenses of 25 million dollars. We plan to use cash from this merger to support clinical, manufacturing and preclinical activities for our development candidates, and for working capital and general corporate purposes. We project that these funds will enable us to achieve multiple key milestones and provide a cash runway into mid-2023. We caution you that this is an estimate only and we cannot assure you that our actual experience will not differ from this estimate.

Upon closing of the business combination, which we anticipate occurring in the first quarter of 2022, the merged company will be named Blade Biotherapeutics, Inc. The company's common stock is expected to be listed on the Nasdaq exchange.

Slide 6 (06:00)

Today, new well-tolerated therapies that provide robust attenuation of disease progression are desperately needed. The disease burden of fibrosis and neurodegeneration is exceedingly high. Some examples include interstitial lung disease, IPF, cardiac fibrosis secondary to hypertension and coronary artery disease. Examples of neurodegenerative diseases that may be targeted by our experimental therapeutics include spinal cerebellar ataxia II, known as Machado-Joseph disease, and Huntington's disease. These are terrible conditions that impact large and growing populations where physicians need new tools to care for their patients.

We at Blade seek to address this with a differentiated pipeline of clinical-stage investigational therapeutics. We believe our product pipeline has the potential to offer a competitive advantage against other experimental therapeutics and the few approved therapeutics.

For fibrosis, our lead asset is cudetaxestat. It binds to the enzyme autotaxin at a non-competitive or allosteric site. Autotaxin is an enzyme that plays a distinct role in fibrosis. Cudetaxestat's non-competitive inhibition of this enzyme offers differentiating preclinical and biochemical characteristics that support the potential for a treatment profile in lung and liver fibrosis and tumors of epithelial origin. We are on track with preparations to initiate a planned phase 2 study in lung fibrosis in the first half of 2022.

Within neurodegeneration, we are pursuing a novel approach that targets the role of dimeric calpains in genetic orphan neurodegenerative conditions. Here, we are advancing a CNS-penetrant calpain inhibitor that is approaching a phase 1 study that is expected to begin in the first half of 2022.

Slide 7 (08:00)

The strategy and science underpinning this combination is informed by biopharma executives with deep experience in the research, development and launch of innovative therapies. We anticipate that the combined company will also be advised by scientific experts with deep experience in fibrosis and neurodegenerative diseases, as well as our experienced combined board of directors.

Slide 8 (08:26)

Underpinning our company and this merger is our pursuit of novel science to understand cell and tissue damage responses resulting from the deposition or buildup of proteins in fibrosis and neurodegeneration. These responses contribute to progressive diseases that, if their root causes are not addressed, may disrupt cells, tissues, and organs with deadly consequences.

Our approach centers on targeting key pathways believed to contribute to disease progression, including autotaxin/LPA and dimeric calpains.

Slide 9 (09:02)

As a result of our compelling research, Blade expects to advance a differentiated pipeline of oral, small-molecule therapies that include a non-competitive autotaxin inhibitor and inhibitors of dimeric calpains designed for potential treatment of lung, liver and cardiac fibrosis, as well as neurodegenerative diseases.

Our lead asset is cudetaxestat, a non-competitive, reversible autotaxin inhibitor that is in clinical development for idiopathic pulmonary fibrosis – or IPF. Cudetaxestat has demonstrated direct anti-fibrotic activity, differentiated preclinical and biochemical characteristics, and showed encouraging safety data to support the potential for a treatment profile in lung and liver fibrosis and oncology. We are confident about our path toward initiation of a planned phase 2 trial in IPF in the first half of 2022. I will talk more about clinical and regulatory steps we have taken to inform the intended path ahead of this clinical development program.

In neurodegeneration, we are developing BLD-2184, a novel CNS-penetrant calpain inhibitor. BLD-2184 is an investigational therapy for inherited poly-Q neurodegenerative conditions such as Huntington's disease, Machado-Joseph disease, and other disorders including Parkinson's disease, Lewy Body dementia and multiple system atrophy.

Importantly, we own global rights to this diversified pipeline of clinical-stage investigational medicines that modulate distinct biological pathways.

Slide 10 (10:42)

I will provide context on the leading program in our pipeline, cudetaxestat, which is a non-competitive autotaxin inhibitor that we are developing for lung fibrosis.

Slide 11 (10:54)

Autotaxin is a secreted lysophospholipase largely responsible for the extracellular production of lysophosphatidic acid – or LPA. Increased autotaxin activity levels have been detected in many inflammatory and fibro-proliferative conditions.

Inhibition of the autotaxin pathway is a clinically validated pathway, which was demonstrated through previous clinical studies of other molecules in IPF, the first fibrotic disease that Blade is researching with cudetaxestat.

Advanced fibrosis is marked by poor outcomes, high morbidity, and high mortality. In IPF, overall expected survival for patients is usually about 3-5 years following diagnosis. It is a terrible disease where

patients essentially suffocate. Despite this, about 30 to 40 percent of patients on currently approved therapies for IPF discontinue treatment due to side effects.

Slide 12 (11:54)

Cudetaxestat is Blade's most advanced development program. This investigational product is differentiated by the way that it binds to autotaxin. Cudetaxestat acts at an allosteric site to reversibly inhibit the enzyme. That's what defines "non-competitive" inhibition.

Cudetaxestat provides distinct chemical and biological activity that separates it from other autotaxin inhibitors.

Slide 13 (12:21)

Based on biochemical and *in vivo* pharmacology studies, cudetaxestat is distinguished by its non-competitive, reversible, and selective inhibition of the enzyme autotaxin. This graphic explains the important distinction.

Since a competitive inhibitor competes with the substrate lysophosphatidylcholine – or LPC – for binding, the activity of a competitive inhibitor can be highly sensitive to substrate concentrations. A competitive inhibitor, therefore, loses potency under conditions of high substrate exposure, such as those found in fibrotic disease tissues.

Results from preclinical studies show that this is not the case with a non-competitive compound. Since cudetaxestat binds at a site not impacted by substrate binding, it inhibits the enzymatic activity regardless of whether or not the substrate is bound to the active site. A non-competitive inhibitor maintains its potency under high LPC concentrations such as those that are found in fibrotic tissues.

To the best of our knowledge, cudetaxestat is the first and the only non-competitive autotaxin inhibitor that is in clinical development at this time.

Slide 14 (13:44)

Autotaxin inhibition by cudetaxestat demonstrates potent and direct anti-fibrotic activity, as shown in preclinical models of fibrosis. Here we see inhibition of key gene markers of myofibroblast activation and differentiation, namely alpha smooth muscle actin and collagen 1A1. This translates into inhibition of assembled collagen, which is a hallmark of advanced fibrosis. Furthermore, consistent with the previously outlined advantages of a non-competitive inhibitor, we see a robust dose response for cudetaxestat, as tested in preclinical fibrotic disease models.

Slide 15 (14:25)

As shown in this preclinical *in vivo* model, twice-daily dosing of cudetaxestat also impacted several profibrotic genes, consistent with the central role that autotaxin-LPA pathway reportedly plays in fibrosis. Therapeutic targeting of IL-6 or CTGF were shown to be clinically beneficial in patients with lung fibrosis through clinical studies of other agents that either are approved or in late-stage clinical development.

Slide 16 (14:58)

Available data from a completed phase 1 clinical study in healthy volunteers showed that cudetaxestat was well tolerated with a supportive clinical safety profile. The pharmacokinetics and pharmacodynamic profile also showed good correlation. At increased doses, we saw robust and sustained reductions in the target engagement biomarker – reduction in LPA-C18-2. We also did not observe any treatment-emergent safety or SAE events at target doses.

Slide 17 (15:32)

We have also completed several *in vivo* and *in vitro* preclinical analyses that support the safety profile of cudetaxestat. In our two-species toxicology program, we did not observe any toxicology signals of concern to date. We have completed several important safety pharmacology studies, two gene toxicity studies, and comparative *in vitro* and *in vivo* CYP profiling against two approved drugs for IPF, pirfenidone and nintedanib. Based on our findings to date, we believe that it will be unlikely for interactions to occur with either of those two drugs.

Following discontinuation of Galapagos' competitive autotaxin inhibitor, known as ziritaxestat or GLPG-1690, we took several stepwise actions to further inform how to appropriately advance our program. This included planning a clinical drug-drug interaction study – or "DDI" study – with the two FDA-approved medications for IPF – nintedanib and pirfenidone, both of which have known safety and tolerability issues.

Slide 18 (16:44)

Ahead of the planned clinical DDI study with nintedanib and pirfenidone, we wanted to assess the potential for any drug-drug interaction in preclinical *in vitro* and *in vivo* assays. Published data reported that nintedanib is a substrate for P-glycoprotein, or "P-gp." P-gp functions as a pump to excrete certain molecules out of cells and tissues. It is essentially a biological barrier that plays a significant role in drug absorption, exposure, and disposition. Inhibition of P-gp at physiologic drug concentrations could dramatically increase the plasma exposure of nintedanib and potentially produce one type of adverse drug-drug interaction.

In a preclinical *in vitro* study in human hepatocyte cells, we found that cudetaxestat is neither a substrate nor an inhibitor of P-gp at concentrations that are physiologically relevant. Conversely, the study showed that ziritaxestat is both a substrate and an inhibitor of P-gp at concentrations that may be achieved *in vivo*.

Slide 19 (17:54)

To evaluate the potential for *in vivo* drug-drug interactions with nintedanib, we conducted a rat PK study. Plasma PK data of nintedanib dosed alone was compared to PK levels achieved when co-administered with either cudetaxestat or with ziritaxestat in the same animals. Results showed that ziritaxestat significantly increased the plasma exposure of nintedanib by 2-3 times, while cudetaxestat coadministration did not increase or change nintedanib exposure.

Slide 20 (18:31)

As these data show, the Cmax of nintedanib increased almost two-fold and the area-under-the-curve increased nearly three-fold when co-administered with ziritaxestat. No significant change in plasma concentration of nintedanib were seen when co-administered with cudetaxestat.

Slide 21 (18:51)

In addition to the P-gp transporter preclinical *in vitro* and in *in vivo* studies, we have taken other actions to support the intended path forward for cudetaxestat. This includes obtaining regulatory feedback from the US FDA to inform a planned phase 2 study in lung fibrosis.

After reviewing our program, the FDA recommended initiating an additional phase 1 study in healthy volunteers to assess whether cudetaxestat affects the pharmacokinetics of the two FDA-approved therapies for IPF, pirfenidone and nintedanib. This planned study in healthy volunteers is anticipated to be completed in the first quarter of 2022. It is intended to evaluate whether there are any significant alterations in PK or tolerability of either cudetaxestat, pirfenidone or nintedanib.

The data from this study are intended to give us guidance necessary to safely co-administer the currently approved IPF therapies to patients who enroll in clinical studies of cudetaxestat in IPF. This phase 1 study should inform how to best proceed into our phase 2 study in IPF, not whether to proceed.

Based on preclinical *in vitro* and *in vivo* studies that we have conducted to look at cudetaxestat and the potential for drug-drug interactions, we believe that there is low risk of potential drug-drug interactions with either nintedanib or pirfenidone.

Slide 22 (20:29)

We believe that our stepwise strategy will serve us well as we work toward final design of our planned phase 2 study in patients with IPF. This 48-week trial is planned to start in the first half of 2022, with the first interim data readout planned for the first half of 2023. We are confident about the anticipated path forward for our lead investigational medicine.

Slide 23 (20:56)

Looking at our planned development programs, in the first half of ’22 we have the potential to advance both programs into the next phase of clinical trials.

We plan to begin a phase 2 study with our lead compound cudetaxestat in patients with lung fibrosis. And, pending completion of the IND application process, we plan to start a phase 1 study of our CNS-penetrant calpain inhibitor BLD-2184 in healthy volunteers.

Slide 24 (21:26)

Over the course of the next 24 months, we have a succession of anticipated milestones in fibrosis and neurodegeneration. We are pleased to have already achieved two milestones on this list. The FDA has activated the IND for cudetaxestat, and one of our phase 1 studies evaluating cudetaxestat was recently completed. We believe that Blade is building momentum, and we are intently focused on executing activities that support these anticipated milestones.

Slide 25 (21:59)

We are confident in the potential offered by our cutting-edge science, differentiated pipeline of clinical-stage investigational medicines, and experienced leadership team. In our view, Blade has the capability to bring potentially life-changing therapeutics to patients with fibrotic and neurodegenerative diseases.

On behalf of Michael Shleifer, the teams at BAC and Blade, thank you for your consideration.

Slide 26 (22:28)

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