Overview

viDA Therapeutics, Inc. (viDA), is a Vancouver, Canada based biotechnology company advancing first-in-class drugs based on inhibitors of the extracellular serine protease, Granzyme B (GzmB), to treat autoimmune and chronic inflammatory age related diseases. The Company is seeking a Series A financing of US$7.0 million to advance our lead drug product, VTI-1002 in gel into the clinic for the treatment of the orphan autoimmune skin disease, Discoid Lupus Erythematosus (DLE). This disease is a severe chronic dermatological disease affecting women five times more than men. and can have a significant impact on quality of life. Based on preclinical studies with the lead compound VTI-1002, the Company is planning to file an Investigational New Drug (IND) application with the FDA within the next 15-17 months.

DLE Program

Currently, there are no products approved for the specific treatment of DLE. Many of the drugs described in the literature are approved for use in other immunological disorders and used off-label for DLE. Current treatments are centered on a regimen of topical and systemic therapies aimed at reducing disease activity through anti-inflammatory mechanisms. Such approaches have had limited efficacy and show significant side effects such as skin atrophy.

Our approach is through a novel mechanism involving extracellular GzmB, an enzyme that degrades extracellular matrix proteins. GzmB-targeted therapeutics is designed to attenuate disease processes that contribute to the disfigurement associated with DLE. By inhibiting the protein degrading activity of extracellular GzmB and preventing extracellular matrix protein degradation, our GzmB inhibitors not only prevent tissue injury but facilitate normal tissue repair and remodeling resulting in accelerated healing and reduced scarring.

VTI-1002 is a first in class drug selectively and potently targeting the GzmB that accumulates in the extracellular space during excessive and/or dysregulated inflammation. By inhibiting extracellular GzmB' pathological activity and preventing extracellular protein degradation, the body can achieve healthy tissue remodeling and repair in DLE lesions. The product will be delivered daily as a topical gel.

Market Opportunity

DLE is a chronic skin eruption that can either be localized or widespread and can cause permanent scarring. The disease mainly affects areas exposed to sunlight, such as the cheeks, nose, ears, upper back, neck and the backs of hands. More common in women of African American, Hispanic and Asian descent, DLE may occur at any age, but most often develops in persons aged 20-40 years. Exacerbation is common with increased sun exposure, particularly in the spring and summer. The condition may persist for years with repeated occurrences and may be chronically debilitating. It is a disease without a cure and there is a continuous need for an effective and safe treatment to prevent permanent disfiguring damage to the skin.

In the US the estimated prevalence of DLE is 135,000 patients with approximately the same number of patients in the European Union (EU). The patient population in both the US and EU will enable viDA to seek an Orphan Drug Designation from both the FDA Office of Orphan Products Development and the European Medicines Agency for VTI-1002. The combined US and EU market is >US$1.0 B (IMShealth.com) with potential additional markets in Latin America and Asia.

Competitive Landscape

Several products with anti-inflammatory activity such as topical corticosteroids, calcineurin inhibitors and anti-malarial drugs may be used as anti-inflammatory treatments off-label for the condition. These current treatment options have limited efficacy and a number of significant side effects including atrophy and steroid dermatitis (topical corticosteroids and calcineurin inhibitors) and retinopathy and cornea deposits (anti-malarial drugs).

The viDA inhibitors are not classified as anti-inflammatory agents. Our GzmB inhibitors are designed to target extracellular GzmB that is significantly elevated in DLE patient lesions. GzmB degrades extracellular matrix proteins contributing to the observed skin damage and impaired tissue healing and/or scarring. This unique approach of targeting an enzyme for which no known extracellular inhibitors have been identified, is differentiated from other approaches that target inflammation.

We anticipate a topical VTI-1002 will be used alone or in combination with current treatment protocols. A therapeutic without the limitations and side effects of current treatment protocols would be a major improvement and provide a significant advancement in the management of the disease.

The Company

viDA has offices on Great Northern Way, Vancouver providing administration, intellectual property, drug discovery and development support. Our research is performed at laboratories at the University of British Columbia. The UBC laboratories consist of multiple scientists researching the role of granzymes in tissue injury, inflammation and disease under the direction of Dr. Granville.

Management

The management team is led by co-founders **Alistair Duncan, BSc CA**, and **David Granville, BSc, PhD, FAHA**. Mr. Duncan, President and CEO, previously with Ernst & Young's Corporate Finance, International Life Sciences group, has since founded three companies involved in gene/cell therapy (technology acquired by GSK), therapeutics (technology acquired by Glenmark Pharmaceuticals) and sustainable bio-jet fuel (in commercialization stage). Dr. Granville, CSO and Professor at University of British Columbia has received multiple recognitions for his research including a Canada Research Chair, Michael Smith Foundation for Health Research Scholar, Canada Top 40 Under 40 and Scholar of the Royal Society of Canada, is a recognized leader in the granzyme field with numerous publications, talks, awards and patents.

viDA is fortunate to have attracted other successful biotech executives to serve in advisory roles:

Dr. Robert Ryan, Chair of viDA board, was co-founder and CEO of Scioderm (acquired by Amicus for US$957-million) that is developing SD-101 for the orphan indication, epidermolysis bullosa, **Dr. Julia Levy**, Board member, was co-founder and CEO of QLT that developed Visudyne generating revenue of US$480 million at its peak and treating over 2 million patients with age-related macular degeneration. **Dr. Marlene Haffner**, Board member, was the Director of the Office of Orphan Products Development at the Food and Drug Administration (FDA) for 20+ years, **Dr. Michael Abrams**, Science Advisory Board Member Chair, was co-founder and CEO of Anormed (acquired by Genzyme for US$580-million) that developed MOZOBIL for a stem cell transplant, **Dr. Ron Nardi,** Dr. Nardi currently serves as Chief Scientific Officer of Scioderm LLC, an Amicus Therapeutics company, focused on the development of an innovative topical treatment for Epidermolysis Bullosa and **Dr. Robert Young**, viDA Science Advisory Board Member, previously Vice President, Chemistry Merck whose team developed the asthma drug, Singulair™.

Intellectual Property

Through a combination of co-ownership and an exclusive license with the University of British Columbia, viDA has an intellectual property portfolio consisting of 50 patents/patent applications (23 issued and 27 pending) including methods of use, composition of matter, formulation and diagnostics around the granzyme technology.

Financing strategy

To date, viDA has raised approximately $10 million (US$8 million) from BDC Venture Capital, private investors and grants ($2.5 million / US$2.0 million) to advance our understanding of the role that extracellular GzmB plays in chronic inflammatory age related and autoimmune diseases and the therapeutic value that can be realized by inhibiting and stopping extracellular GzmB's pathological activity.

We are currently seeking US$7.0 million to fund our lead candidate, VTI-1002 in a gel topical formulation through the completion of an IND application with the FDA, a Phase I clinical trial and a Phase 2 clinical trial and to obtain an Orphan Drug Status to treat DLE.

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