viDA THERAPEUTICS INC.
Building a portfolio of proprietary first-in-class drugs for the treatment of diseases involving fibrosis, inflammation and aging

STATUS
Developing an inhibitor platform targeting granzymes for the treatment of chronic inflammatory, connective tissue and autoimmune diseases; Lead development program in preclinical stage for dermatology indication

MULTIPLE APPLICATIONS
- Dermatological
- Musculoskeletal
- Respiratory
- Cardiovascular
- Neurological
- Cancer
- Autoimmune

MILESTONES
- First follow-on series of inhibitors – 1Q13
- Execute proof of concept in additional disease models – 4Q13
- Complete V2248 pharm/tox studies – 2Q14
- Phase I start in dermatology indication – 3Q14

INTELLECTUAL PROPERTY
Patent portfolio filed covering age-related degenerative processes, wound healing, cardiovascular disease and other inflammatory conditions with granzyme inhibitors

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Overview
viDA Therapeutics, Inc. (viDA) is a platform-based, biotechnology company. We are developing first in class therapeutics, across a broad range of diseases, capitalizing on our recent discovery of a new mechanism of action for a well characterized protease target, Granzyme B (GzmB). This is emerging science potentially leading to new treatments for diseases of fibrosis, inflammation and aging and is pivotal in understanding the underlying causes of diseases affecting vasculature, skin, musculoskeletal and potentially neurological systems. GzmB is one of a family of five serine proteases now emerging as key targets for drug development given recent discoveries of their extracellular actions. We are developing a suite of granzyme inhibitors designed for multiple routes of administration to treat chronic and orphan skin conditions, fibrosis, rheumatoid arthritis, and cardiovascular disease.

Currently, our focus is specifically on extracellular GzmB and its role in conditions involving inflammation and tissue damage. Extracellular GzmB actively participates in degradation of key components of the body's extracellular matrix, which acts as a scaffold for cell binding and provides essential structural integrity for proper function of tissues and organs. Our aim is to demonstrate the clinical potential of inhibiting extracellular GzmB in dermatology first, before attempting clinical development in other indications.

Our lead compound, V2248, is at preclinical stage and is intended as a topical treatment for hypertrophic scarring, keloids, and chronic and/or diabetic wounds. V2248 exhibits a high degree of specificity and selectivity for extracellular GzmB and is exhibiting compelling drug-like properties in our preclinical studies. We have shown that V2248 inhibits human GzmB in a dose dependent manner in wound fluids and plasma and is highly selective for this protease. The compound does not induce skin or wound irritation upon daily application in a carrier formulation for up to thirty-two days, has attractive drug-like properties for topical application, and is showing excellent safety/ADME characteristics based on early screens and pig studies.

Granzyme B – An Ideal Target, Multiple Indications
An abundance of data has emerged in the literature demonstrating a correlation between elevated GzmB in bodily fluids and increased severity of many chronic diseases of inflammation and/or aging. GzmB is not naturally inhibited in the extracellular milieu. As such, levels and consequently protein degradation tend to increase substantially in disease states without any apparent restraint. High levels of GzmB result in inflammation and/or connective tissue destruction. We have elucidated the mechanism of action in animal studies of aortic aneurysm and wounds and, in some cases, have validated GzmB as a key contributor in the pathogenesis of certain diseases in which high extracellular GzmB levels are prevalent. Studies using inhibitors and knockout approaches demonstrate that GzmB inhibition does not suppress immunity.

Our animal data supports potential systemic administration of large molecule (proteins, Mabs) GzmB inhibitors: Intravenous injection of a novel biologic inhibitor of GzmB or a neutralizing anti-GzmB antibody attenuated vascular injury and rupture in preclinical models of aortic aneurysm. Increased survival was also observed when extracellular GzmB was inhibited in these models.

Inhibitor Discovery
To develop potential treatments, our drug discovery program is focused on the generation of novel, small molecule GzmB inhibitors, however, we are examining

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biological GzmB inhibitors for certain indications. These innovative molecules will have specific properties amenable to treatments of diseases requiring different routes of administration such as IV (or direct injection) for treatment of RA or IV for the dermatological orphan diseases, SJS and TENS. The small and large molecule drug discovery programs will expand to target the rest of the granzyme family over time.

Clinical Proof of Concept

Our aim is to demonstrate a clinical benefit by inhibiting extracellular GzmB in the area of dermatology first, before attempting clinical development in other indications. Dermatology offers both an economical and a timeline advantage in moving our lead program into a clinical pathway for drug development and it provides the opportunity to explore several skin conditions involving chronic injury, inflammation and repair.

We have demonstrated, in models of vascular and skin injury, that inhibition of extracellular GzmB promotes tissue repair by preventing the degradation of key extracellular matrix proteins that are essential for wound repair and tissue remodeling. Histological studies determined that one of the key substrates that was degraded in the skin by GzmB was decorin. Decorin is the most abundant proteoglycan in the skin and is responsible for the tensile strength, thick bundle formation and organization of collagen. The absence of decorin in skin is observed in a number of skin diseases and experimental studies have shown that genetically knocking out decorin leads to reduced skin tensile strength due to abberrant collagen organization. Loss of decorin results in a decrease in the size and organization of collagen in the skin, influencing the structural integrity of the tissue. Accumulating evidence suggests that elevated GzmB levels, as a consequence of infection, environmental exposure/injury and/or age can augment skin injury and impair skin repair.

Our data demonstrate dose-dependent and selective inhibition of GzmB in pig and human biological fluids (wound fluid, plasma) and improved wound healing in mouse models as well as safety in pigs. Further proof of concept studies is underway to assess the efficacy of V2248 in dermatological indications. Following these studies, we intend to advance V2248 into toxicological and clinical development.

Chronic Inflammation and Age Related Disease

Inflammation is a physiological process that normally helps us fight infection and aids in tissue repair. Dysfunctional immune responses, however, contribute to the development and progression of several common chronic diseases including asthma, cardiovascular disease, arthritis, diabetes, non-healing skin ulcers and cancer. Approximately 133 million Americans – 45% of the US population – have at least one chronic disease. In the U.S., total spending on public and private health care amounted to approximately $2 trillion during 2005, of which more than 75% went toward treatment of chronic disease. Continued spending growth will be driven in part by the greater prevalence of chronic illnesses, and the longer life expectancy of the population.

Funding Strategy

viDA has raised $4.5 million to date, with the last round led by BDC Capital. We have also secured non-dilutive, peer reviewed grant funding of over $1.2 million. We are seeking additional funding to support and accelerate the ongoing development of V2248 and to expand our suite of granzyme inhibitors.